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The Effect of Photoperiod, Ergocryptine Injection and Melatonin Feeding on Reproduction in the Ewe

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THE EFFECT OF PHOTOPERIOD, ERGOCRYPTINE INJECTION AND MELATONIN FEEDING ON REPRODUCTION IN THE EWE

BY
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REPRODUCTION IN THE EWE

This thesis is approved as a creditable and independent investigation by a candidate for the degree, Master of Science, and is acceptable for meeting the thesis requirements for this degree. Acceptance of this thesis does not imply that the conclusions reached by the candidate are necessarily the conclusions of the major department.

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INTRODUCTION

Seasonal breeding in ewes limits productivity and causes an unsteady supply of lambs to the market place. A successful and practical method of causing a continuous breeding season in ewes is needed to stabilize lamb supplies, farm labor and utilization of facilities. Causing ewes to exhibit fertile cycles during the normal summer anestrous period would be a means of extending the lambing season.

In an effort to do this, alteration of the light: dark cycle and various chemical compounds were studied in anestrous Targhee and Finnsheep x Targhee ewes. Ergocryptine was used because it has been shown to drastically lower plasma prolactin levels. Prolactin levels are high in summer months and are thought by some to be the cause of summer anestrus in ewes.

Melatonin was tested in an attempt to alter normal summer anestrus. Melatonin is a substance secreted by the pineal gland in response to darkness. As night length increases, the duration of the high melatonin levels increase. As night length decreases, melatonin duration also decreases. The characteristic pattern of melatonin secretion is thought to "signal" the ewe's system to initiate reproductive activity.

REVIEW OF LITERATURE

Ever since early time man has realized that there is a certain cyclicity in nature. Ecclesiastes 3:1-2 attests to this fact; "For everything there is a season...a time to be born and a time to die." This cyclicity is often seen in the form of seasonality of reproduction in certain animal species. Seasonality of reproduction is necessary for survival of some species, especially in those that rely on adequate amounts of high quality vegetation for late fetal growth and lactation, and on mild temperatures for neonatal survival (Turek and Campbell, 1979).

One of the most intriguing aspects of reproduction is the reversible fertility governing seasonal breeding. Sheep are one of the many species that exhibit a circannual rhythm in reproduction, because they start estrous cycles in fall and winter months, and become anestrus in spring and summer months. Seasonal changes appear to control this (Yeates, 1949). Ewes seem to be more completely seasonal than are rams because only very limited ovarian activity, fewer follicles, and no ovulation occurs in ewes, although follicular size remains unchanged (Kammlade et al., 1952). Most rams will produce viable semen throughout all seasons, however, Lincoln and Short (1980) and Almeida and Lincoln (1984) reported that Soay rams showed cyclicity in

testicular size and semen quality associated with season of the year or with changes in photoperiod.

A major restriction to intensive, year round lamb production is the seasonal anestrus exhibited by ewes. Environmental conditions, genetics, and hormonal status are three of the factors that may cue the ewe's system to initiate or cease reproductive activity, and are the three main factors that researchers have tried to manipulate in order to achieve out of season breeding.

Environmental Conditions

Many environmental factors have been implicated in causing the yearly cycle seen in sheep, including nutrition (Ducker and Boyd, 1974; Rhind et al., 1980) and temperature (Dutt and Bush, 1955; Godley et al., 1966), but the factor seeming to exhibit the most control is photoperiod, i.e. a seasonal variation in the length of daylight versus darkness (Yeates, 1949; Hafez, 1951; Legan and Karsch, 1980). Photoperiod, in turn, appears to affect hormonal secretion (Bittman et al., 1983b).

Season and Photoperiod

Ewes respond to increasing daylength by cessation of estrous activity (Hafez, 1951; Ducker and Bowman, 1970a), but to decreasing daylength by initiation of estrous cyclicity (Yeates, 1949; Hafez, 1951). However, the time of year that treatment is applied is important.

For example, Ducker and Bowman (1970b) reported that ewes kept in a treatment of 8 h light and 16 h dark (8L:16D) starting July 1 exhibited estrus an average of 59.5 d later, but ewes started on a similar lighting regime on April 24 took 87.0 d to reach estrus. They hypothesized that this difference is due to either an internal rhythm or to temperature changes. Others report that anywhere from 27 to 80 d on this lighting schedule are necessary to initiate reproductive activity (Hafez, 1952; Vesely, 1978). All lighting treatments of this type appear to have a lag time associated with them that must be overcome, but time to estrus can be shortened by certain photoperiodic treatments (Ducker et al., 1969).

A hypothesis for the differences seen in lag times during different parts of the year is that the ewe becomes photorefractory, and so late in the breeding season or early in the anestrous season she will respond less readily to short days (Worthy and Haresign, 1983). Speedy and Owen (1975) noted that they could not prolong the breeding season with light if it was applied after the shortest day of the year (December 22). It was determined by Robinson and Karsch (1984) that transition into the breeding season occurred when daylength was longer (14.0 h light) than the daylength at the onset of anestrus (11.5 h). From this they assumed that ewes become

photorefractory. To test this hypothesis ewes were kept in 10 h light or in natural daylight; both groups reached anestrus at approximately the same time. These authors attributed photorefractoriness to a disruption of the post-pineal processing of the photoperiod message (to be discussed later).

Some studies indicate that a slow decrease in daylength is necessary for the initiation of the breeding season (Dustan, 1977), but others indicate that an abrupt change to short days is adequate (Hart, 1950; Hafez, 1952), and can decrease time to first estrus (Ducker et al, 1970). Type of light used, artificial or natural, show no differences (Newton and Betts, 1972). It appears that it is only the ratio of light to dark that must fall below a certain level before reproductive activity can be initiated.

All photoperiodic information to the sheep is received by the eye, however, Legan and Karsch (1983) discovered that after blinding, ewes still showed circannual alterations between cyclicity and anestrus. Ewes kept under constant light showed seasonality (Radford, 1969), as did ewes kept in equinoctial lighting, i.e. 12L:12D (Thwaites, 1965). A more sporadic season, and large individual differences were noted between ewes in these studies, probably due to an inherent rhythm in the animal. It can be assumed from these experiments that

endogenous circannual rhythms in reproductive activity are not solely dependent on seasonal changes in photoperiod, but rather are entrained to photoperiod.

An endogenous diurnal rhythm also appears to exist in sheep. Almeida and Lincoln (1982) found that rams kept in 8L:16D or in 8L:40D (multiples of 24 h) had an increase in testicular development and skin flush, and also had a 24 h periodicity in prolactin and melatonin concentrations. Rams kept in 8L:28D (not a 24 h rhythm) exhibited no constant pattern in either testicular and skin changes, or in hormonal pattern.

Genetics

Genetics also play a role in determining the length and onset of the breeding season in sheep (Hafez, 1952). The majority of sheep are short day breeders although some breeds, such as the Dorset Horn (breeding season ~ 223 d) are more capable of year round breeding (Hafez, 1952). Individuals of Suffolk breeding are more variable in their anestrous period, starting some time in January to early April and ending in late August; Hampshires generally become anestrus in February or March and begin to cycle again in late August (Bellinger and Mendel, 1974). The Finnsheep's breeding season occurs from October to May (Wheeler and Land, 1977). Finnsheep x Targhee crossbred ewes had a 14 d later and a more variable onset of estrus

in the fall than did Targhee ewes, but also exhibited fewer missed estrous cycles once the breeding season was established (Meyer and Bradford, 1973). Although individuals within a breed show wide variation, genetic make up of the animal can influence the effect of photoperiod on that particular animal.

Hormonal Factors

Gonadotropins and Estradiol

The effects of photoperiod on reproduction in sheep are mediated by changes in the pattern of luteinizing hormone (LH) release. During the normal ovarian cycle of the breeding season LH patterns reflect two separate regulatory systems, a tonic system which produces low pulsatile discharges of LH, and a surge system which produces the massive pre-ovulatory LH release (Scaramuzzi and Baird, 1977; Legan and Karsch, 1979). This LH surge precedes ovulation by approximately 24 h, and is the cause of behavioral estrus because of its role in estradiol production by the enlarging Graffian follicle (Legan and Karsch, 1979). At ovulation the Graffian follicle releases the ovum. Ovulation, in turn, leads to corpus luteum (CL) formation, and progesterone production. LH then decreases. It may, in fact, be that high progesterone concentration exerts the dominant inhibition of tonic LH secretion during the estrous cycle of the ewe (Legan and Karsch, 1979).

(Progesterone, of course, plays no role in the control of LH secretion at anestrus, as its concentration is very low at this time.)

After a 12 to 14-d luteal phase, progesterone concentration decreases, CL regression occurs, and the resultant rise in serum LH stimulates growth of new ovarian follicles. These follicles produce estrogen of sufficient quantity to initiate a pre-ovulatory surge of LH and the cycle repeats itself (Legan and Karsch, 1979).

Little work has been done on the role of follicle stimulating hormone (FSH) in this sequence of events. Some work that was done by Platt et al. (1983) noted a depression of FSH output, but not LH output in a short day photoperiod in ovariectomized ewes given estradiol.

Most of the essential components of the hypothalamo-hypophyseal-ovarian axis just presented remain functional in the ewe as days lengthen and yet anestrus occurs (Legan and Karsch, 1979; Karsch et al., 1980). The major cause of this phenomenon is a change in the sensitivity of LH release to estradiol. In short days (the breeding season) LH has a low response to estradiol inhibition, yet in long days (the anestrous season) estradiol depresses LH levels (Legan et al., 1977; Karsch et al., 1980; Goodman et al., 1982). It has been shown that 3 pg/ml or more of estradiol (with progesterone

priming) will induce estrus in a ewe, no matter what the season (Goodman et al., 1981). In anestrous periods, 1-3 pg/ml of estradiol suppressed LH to low levels, however, in the breeding season even 10 pg/ml of estradiol was unable to cause LH inhibition (Goodman et al., 1981). Goodman et al. (1981) suggested that estrogen is a more potent inhibitor of LH secretion during anestrus because it gains the capacity to suppress the frequency of gonadotropin releasing hormone (GnRH) discharges from the hypothalmus. This was determined because response of the pituitary to GnRH injections was not decreased during anestrus.

Luteinizing hormone amplitude and pulse frequency are both affected by estradiol negative feedback. Estradiol has a limited capacity to decrease LH amplitude in the breeding season, but has no effect on LH pulsing. However, in the anestrous season, LH pulses are depressed although basal levels may not be (Karsch et al., 1980; Goodman et al., 1982). There are also seasonal changes in the pulsatile nature of LH secretion that are independent of steriod (either estrogen or progesterone) feedback, and these may contribute to the low LH pulse frequency seen during anestrus. These pulsatile discharges are not sustained, however, and so ovulation can not occur during anestrus (Legan and Karsch, 1979).

Little is known about FSH during the transition to anestrus or during anestrus. Kammlade et al. (1952)

hypothesized that anestrus may be due to an imbalance of FSH and LH. Follicle stimulating hormone does not exhibit as large a relative drop in concentration as does LH under long photoperiods and will cause follicular development during anestrus (Kammlade et al., 1952). However, estrogen will not be produced by the follicles without adequate LH concentration, as stated earlier.

It appears that estradiol can be considered the organizer of the seasonal cycles seen in sheep since the absence of estrous cycles during anestrus is a consequence of the negative feedback loop between LH and estradiol, which prevents the pre-ovulatory LH surge. To accomplish out of season breeding, this loop must be opened so that the sequence of events leading to ovulation can occur.

Prolactin

What causes the change in LH inhibition by estradiol is not known, however, it may be partially controlled through prolactin.

Changes in prolactin release parallel changes in photoperiod (Webster and Haresign, 1983). Prolactin is characteristically high during anestrus and low during the breeding season (Walton et al., 1980). It may be that high levels of prolactin reduce the release of LH in response to estradiol (Lamming, 1974; Walton et al., 1980). A fall in prolactin concentration occurs in ewes at the transition

from anestrus to the breeding season (Walton et al., 1977). The return to estrous activity may be brought on by the removal of the anti-gonadotropic effects exerted by high levels of prolactin in the blood at anestrus (Walton et al., 1977). Rhind and coworkers (1980) noted reduced fertility in ewes mated in March or July versus those mated in December. Prolactin values for these ewes averaged 200 ng/ml and 35 ng/ml, respectively. Therefore, it was suggested that prolactin is deterimental to fertility either by interfering with LH release or by decreasing luteal function. McNeilly et al. (1982) discovered that high concentrations of prolactin may even effect steroidogenesis in an experiment involving hyperprolactinemia in post-partum women.

Some researchers, however, have concluded that prolactin does not control seasonal anestrus in sheep, but its increase is just a coincidental phenomenon (Schanbacher, 1980; Webster and Haresign, 1983; Worthy and Haresign, 1983). Worthy and Haresign (1983) noted that ewes kept in 8L:16D for extended periods of time become anestrus, yet still had significantly lower average serum prolactin levels than their counterparts in natural daylight during the transition to anestrus.

An ergot alkaloid, 2-bromo-α-ergocryptine (ergo), known to depress serum prolactin in goats (Hart, 1973) and sheep (Niswender, 1974) to levels below .5 ng/ml has also

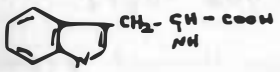
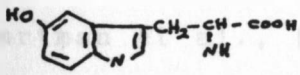
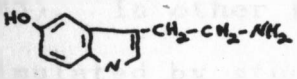
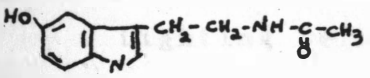
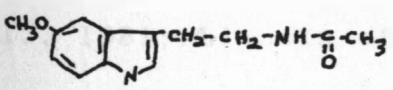
been used to test prolactin's effects on anestrus. Dopamine is the prolactin inhibiting factor, and ergo is a dopamine receptor agonist (Land et al., 1980). Since ergo acts similarly to dopamine, prolactin secretion is inhibited when this drug is used. Ergo did not effect LH, FSH, or progesterone level, nor did it effect luteal function or estrus length, however, prolactin levels were significantly decreased (Niswender, 1974). Ewes given ergo in natural daylight averaged 16.1 d longer to conception than ewes treated with 8L:16D (Schanbacher, 1980). Land and coworkers (1980) gave ewes ergo for 12 d. This treatment had no effect on the proportion of ewes discharging LH or ovulating in response to estradiol injections when compared to control ewes in normal summer anestrus. It, therefore, appears that prolactin plays only a minor role in controlling the seasonality of ovine reproduction.

Melatonin

It has been proposed that photoperiod signals are perceived by retinal photoreceptors and transmitted first to the suprachiasmatic nuclei by way of the retino-hypothalamic tract, to the paraventricular nucleus, along an unknown pathway to the superior cervical ganglia, and then to the pineal gland. Finally, signals are transmitted from the pineal, via a humoral mediator, melatonin, to the hypothalmus (Turek and Campbell, 1979).

Melatonin is a tryptophan derivative. High tryptophan in the diet, however, will not increase serum melatonin levels (Kennaway et al. 1978), although injections of 5-hydroxy tryptophan will (Namboodiri, 1983). 5-hydroxy tryptophan is an intermediate between tryptophan and serotonin in melatonin synthesis (Axelrod, 1971). See Figure 1.

Figure 1. The synthesis of melatonin in the pineal gland. (Adapted from Hafez, 1980.)

COMPOUND		PINEAL CONCENTRATION IN RELATION TO LIGHT	
Chemical Structure	Name	Time of Day LIGHT	DARK
	tryptophan	no relationship	
	← tryptophan hydroxylase		
	5-hydroxy trytophan	probably no relationship	
	← aromatic amino acid decarboxylase		
	serotonin	↑	↓
	← serotonin-N-acetyl-transferase	↓	↑
	N-acetyl-serotonin	↓	↑
	← hydroxy indole-O-methyl transferase	↓	↑
	melatonin	↓	↑

This system is a conversion of neural input into an endocrine output, that is, sympathetic nerves release norepinephrine at their end terminals on pineal cells in a rhythmic fashion that parallels light and dark cycles (Tamarkin et al., 1985). This neurotransmittor is bound by membrane β-aderinergic receptors, the system is activated, formation of serotonin-N-acetyl-transferase occurs, and melatonin formation follows (Tamarkin et al., 1985). How melatonin affects the hypothalmus, however, is still unclear.

It should be noted in Figure 1 that lighting affects not only synthesis of melatonin and its intermediates, but also synthesis of the various enzymes in the pathway (Wurtman et al., 1963).

Light may stimulate the pineal by several different pathways depending on species. In rodents, light can effect the pineal directly by entering through the skull (Wurtman et al., 1963). In sheep, the eye, through the retina and optic nerve, is responsible (Legan and Karsch, 1983). In other species, the pineal may be indirectly stimulated by other light sensitive hormones (Wurtman, 1963).

Lighting serves a two-fold role in pineal metabolism. The first role is a suppressive one in that melatonin is not synthesized in periods of light (Arendt et al., 1981; Lincoln et al., 1981; Lincoln, 1983), and serum

levels of melatonin approach zero at this time. The second function of light in relation to the pineal is to entrain or synchronize pineal rhythm with the environment (Bittman et al., 1983a; Kennaway et al.,1983; Tamarkin et al., 1985). In darkness, serum melatonin levels can reach 2000 pg/ml in some ewes (Rollag et al, 1978). There appears to be some breed differences. Rollag and others (1978) found that Corriedale cross ewes averaged 10-100 pg/ml in light, while Lincoln cross ewes averaged 200-400 pg/ml in the same conditions. Levels rose 2-10 times in darkness.

Photorefractoriness is the physiological state in which an animal is temporarily incapable of responding to a particular light cycle that had previously altered neuroendocrine gonadal activity (Turek and Campbell, 1979). Mammals may also become refractory to exogenous melatonin (Bittman, 1978; Nett and Niswender, 1982) and possibly to endogenous melatonin (this may be what mediates photorefractoriness). In rams exposed constantly to either inhibitory or stimulatory photoperiod alternating periods of gonadal involution and redevelopment were observed (Almeida and Lincoln, 1984). In ewes, photorefractoriness to short days may be characterized by shortened periods of high LH and estradiol concentrations prior to ovulation, and no pre-ovulatory LH surge or occurrence of estrus (Karsch et al., 1980).

Almeida and Lincoln (1984) suggest that there is a loss of coordination between the secretion of melatonin and the light-dark cycle during photorefractoriness, therefore, the circadian control of melatonin secretion is disturbed. Their research showed that when rams were maintained for a prolonged time under fixed photoperiod there was a spontaneous change in the daily temporal pattern of melatonin secretion. Peaks in melatonin occurred at various times of the day, and were not confined to periods of darkness. This occurred after 25 wk of exposure to the same photoperiod.

Possible mechanisms involving melatonin which account for photorefractoriness may be that receptors for melatonin are gradually depleted (Nett and Niswender, 1982), patterns of melatonin release are changed (Almeida and Lincoln, 1984), or the response of the hypothalamic-pituitary-gonadal axis to melatonin may in some way be modified with exposure to unchanging photoperiod (Almeida and Lincoln, 1984).

The central actions of melatonin on reproduction may be mediated through the hypothalamic suprachiasmatic nuclei, the body's "biological clock" (Lincoln, 1979; Tamarkin et al.,1985). Melatonin does not appear to be either pro- or anti-gonadal in sheep, but is more of a control mechanism (Bittman and Karsch, 1984; Karsch et al., 1984). Melatonin may control the sensitivity of tonic

LH secretion to estradiol (Bittman et al., 1983b; Yellon et al., 1985). An increase in melatonin can cause an increase in LH , and as the animal becomes photorefractory LH again declines (Bittman and Karsch, 1984; Karsch et al., 1984). Stage of the cycle does not affect melatonin concentration (Rollag et al., 1978; Seamark et al., 1981).

Proposed mechanisms by which melatonin might control hypothalamic secretions via the GnRH-containing neurons are: 1) alterations of the electrical activity of the neuron, 2) impairment of the contractile processes involved in the axonal transport of GnRH-containing granules, or 3) changes in the synthesis and secretion of the catecholamines, monoamines, and prostaglandins, all of which are believed to regulate release of GnRH (Tamarkin et al., 1985).

Melatonin concentrations change almost immediately with changes in photoperiod, and yet, time to initiation of reproductive capabilities have a lag time of several weeks. Some hypotheses as to why this occurs are: 1) there may be changes in the neuronal structure that takes time to complete, 2) changes in the rate of synthesis or degradation of GnRH may occur, or 3) there may be a melatonin mediated change in the processing of steroids (Bittman et al., 1983a).

With this basic research done, the next step is to determine if the ewe's normal annual cycle can be changed by using melatonin treatment. Melatonin has been injected (Kennaway and Seamark, 1980; Nett and Niswender, 1982), implanted (Kennaway et al., 1982b), and fed (Arendt et al., 1983; Symons et al., 1983), at levels of 2.0 to 2.5 mg, all with equal success in raising serum melatonin levels to those normally seen in darkness. Melatonin treatment is also seen to lower prolactin levels (Kennaway et al., 1982b; Symons et al., 1983) and to initiate estral activity in anestrous ewes after a lag period (Kennaway et al., 1982a; Nett and Niswender, 1982; Arendt et al., 1983).

It appears that exogenous melatonin treatment can hasten the onset of the breeding season in the fall or extend it in the spring. The main question that researchers need yet to answer is: How does melatonin control secretions from the hypothalmus?

Opiod Peptides

The role of endogenous opiod peptides in the control of seasonal reproduction is now being studied. It appears that the opiods play some role, yet many questions about them are still unanswered.

Endogenous opiods are seen to effect LH and prolactin concentrations in ovariectomized ewes (Schillo et al., 1985). It may be that endogenous opiod peptides

exhibit some type of control over the steroid independent system of LH control (Schillo et al., 1985). Continued research is needed on these compounds to determine their effects.

The research presented herein is designed to give insight into a practical way to control seasonal breeding in sheep, as well as to discern part of the possible mechanism that controls this seasonal breeding.

MATERIALS AND METHODS

The study reported herein examined the effects of artificial photoperiod and various chemical compounds to stimulate the onset of estrous activity in anestrous ewes.

Trial 1

Fifty Finnsheep x Targhee crossbred ewes, aged 2 to 5 yr, were allotted within age to one of five treatment groups. Treatments were: 1) ewes serving as controls and maintained in natural daylight (ND), 2) ewes housed inside, exposed only to artificial light at a rate of 8 h light and 16 h dark (8L:16D), 3) ewes in natural daylight and receiving 2.0 mg 2-bromo-α-ergocryptine (ergo, Sigma Co.) per ewe injected im twice weekly (ND+ergo), 4) ewes in artificial lighting and receiving ergo in a similiar manner to those in treatment 3 (8L:16D+ergo), and 5) ewes receiving artificial lighting which decreased one h/wk over an 8 wk period and then held at a constant 16 h dark for the remaining 7 wk (16L:8D-8L:16D). Treatments 1 and 2 also each contained ten, 9-yr old Targhee ewes to compare breed effect.

Prior to the start of the experiment, all animals had their feet trimmed and necks shorn to facilitate venipuncture, and were paint branded. The experiment started May 16, 1983 and terminated on August 25, 1983.

Five semen-tested Suffolk rams were used in the experiment, two for ewes in ND, two for ewes in 8L:16D treatments, and one for ewes in the 16L:8D-8L:16D treatment. An SPE Electro Ejaculator was used for semen collection. Semen was evaluated for motility and concentration using the hanging drop technique. On May 27, the ram allotted to ewes in the 16L:8D-8L:16D treatment was taken out to be treated for illness and a different Suffolk ram was introduced into the group. On June 2 the original ram was returned to this treatment group. On July 6 and August 1 rams were rotated between treatments in artificial lighting.

Ewes were exposed to rams continuously. Rams were painted with a mixture of wool paint and lithium grease twice daily, just cranial to the sheath. Breeding marks were recorded at this time and rated on a scale of good, fair, poor, and rape. A mark directly over the tail head represented a good mark. A mark less than 10 cm to either side of the tail head, but not directly over it was considered a fair mark. A poor mark constituted one more than 10 cm to either side of the tail head, but still in the hind saddle. A rape mark was elsewhere on the body. Grease color was changed periodically.

Sheep were fed 1.8 kg pellets (75% sun-cured alfalfa, IFN 1-00-023; 25% corn cob, IFN 1-28-234) per head daily until May 20 when the amount fed was increased to 2.3

kg per head daily. Sheep were also fed .23 kg rolled corn (IFN 4-28-238) per head daily. On June 21 corn was decreased to .11 kg per head daily and on August 1 corn was dropped from the diet because monthly weighings indicated excessive weight gains. The rams received an additional .23 kg of corn per head daily. A mixture of 50% trace mineralized salt and 50% dicalcium phosphate was available free-choice. Sheep were weighed every 28 d to monitor weight change.

Treatments 2 and 4 were housed together in a temperature and light controlled room (Room A). Treatment 5 was housed in a separate, comparable room (Room B). Lights came on at 0730 h. Light was removed from the end of the light cycle for those ewes in treatment 5 (16L:8D-8L:16D).

Temperature was maintained at 22C$\pm$2C by heating and cooling systems within the building. The cooling system malfunctioned on one occasion for approximately 24 h at which time fans were used to aid in cooling. Temperature increased at this time to 32C.

Room A allowed for 1.1 m^2 pen space per animal. The floor consisted of two 2.43 m wide slotted areas with a 1.52 m wide center concrete alley. Water was supplied via automatic bowl fountains. Room B allowed for 1.9 m^2 pen space per animal. These floors were solid concrete except

for a 45 cm wide grated gutter along the entire length of one wall. Floors were scraped daily and the pit and gutter flushed 3 times per wk.

Artificial light sources in each room were provided by 660 watt cool white fluorescent lights located approximately 220 cm above the ewe's eye level. Light schedules were controlled by Intermatic time switches.

Treatment 1 and 3 ewes were kept outside without shelter in a pen providing 3.5 m^2 per animal. Water was provided in a stock tank. The pen area was dirt, shaded by elm trees.

Ewes in groups 2 and 4 were given injections twice weekly at 0730 h. Injections of 2.0 mg of 2-bromo-α-ergocryptine (ergo) per ml of carrier (60% ethanol, 40% saline) were administered im in the gluteal region. The drug was stored in 100 cc bottles at 4C between bleedings.

A 10 ml blood sample was collected from each ewe twice weekly between 0700 h (ND treatments) and 0800 h (artificial light treatments). Blood samples were collected in vacutainer tubes and labelled with ewe number. Blood was allowed to sit for approximately 30 min at room temperature after bleeding and was then centrifuged (International centrifuge, size 1, Model SBV) in a walk-in cooler, for 30 min at 2500 rpm. Serum was then placed in tubes for storage at -24C. Aliquots were also removed at this time for future prolactin assays.

The experiment terminated on August 25, 1983. Subsequent to this, ewes were exposed to a Columbia clean-up ram. At lambing, ewe number, date and time of birth, birth weight, sex of lambs, type of birth, and breed of sire were recorded.

Progesterone assays were determined on weekly bleedings. Progesterone assay was a solid-phase radioimmunoassay technique. The antibody was immobilized on the wall of a polypropylene tube as prepared by Daignostic Products Corporation, Los Angeles, California. Progesterone was labelled with sodium [^{125}I] iodide with high specific activity and total counts of approximately 90,000 cpm at the time of iodination. Maximum binding was 40-50%. Separation of the bound and free fractions and termination of competition was accomplished by decanting the supernatant after 3 h incubation at room temperature. The bound fraction remained in the tube.

The antibody was checked for crossreactivity to other compounds. Crossreactivity was found to be less than .5%. Sensitivity of the assay was determined to be .05 ng/ml. The standard curve was linear between .05 ng/ml and 100 ng/ml. Increasing volumes of steer serum paralleled the standard curve.

An aliquot of 300 ul was used for samples. Steer serum spiked with 8 ng/ml progesterone served as a check

for recovery. Recovery was found to be 104%. Assay values were calculated by the log-logit method. Intra- and interassay coefficients were determined with the use of pooled steer serum and spiked steer serum. Coefficients of variation for intraassay coefficients was .13, and for interassay coefficients was .25.

Prolactin assays were run for each bleeding. Prolactin concentrations were determined by the double antibody radioimmunoassay procedure described by Schanbacher and Ford (1979) without modification. Purified antigen and antibody were obtained from the National Institute of Arthritis, Diabetes, and Digestive and Kidney Diseases (NIADDK).

Statistical analysis was performed on normally distributed data by least squares procedures (Steel and Torrie, 1980). Waller Duncan K-ratio T-Tests were used to separate means for factors having significant F values (SAS, 1982). Binomial data were tested using Chi-square analysis. Independent Chi-square values were determined to identify significant treatment effects. Correlations between hormone values within week were determined using SAS (1982). A probability level of less than .05 was considered the maximum level at which significance was accepted unless otherwise indicated. Analysis of variance and Chi-square values are shown in appendix tables.

Trial 2

Sixty Finnsheep x Targhee ewes, aged 2 to 6 yr, were allotted within age to one of four treatment groups. Treatments were: 1) ewes in natural daylight (ND), 2) ewes in artificial lighting, treated only with 8 h light, 16 h dark (8L:16D), 3) ewes in natural daylight and receiving 3.5 mg melatonin fed per head daily (ND+mel), and 4) ewes in 8L:16D and fed 3.5 mg melatonin per head daily (8L:16D+mel).

Ewes were prepared for the study similarly to Trial 1. The trial was initiated June 1, 1984 and terminated August 24, 1984.

Four intact Suffolk rams were used in the trial. Semen testing, greasing, and reading breeding marks were done similarly to that in Trial 1, with the exception that rams were greased and breeding marks read only one time/d. Grease color was changed every 17 d. The rams were rotated daily within ND treatments or within 8L:16D treatments. That is, the ram in treatment 1 was rotated with the ram in treatment 3, and the ram in treatment 2 was rotated with that in treatment 4.

Sheep were fed 2.3 kg pellets (74.5% corn cob, IFN 1-28-234; 24.5% sun-cured alfalfa, IFN 1-00-023; 1% molasses, IFN 4-04-696) per head daily. Ewes were fed .11 kg rolled corn (IFN 4-28-238) per head daily. Molasses was added to the corn for binding and dust reduction.

Melatonin (3.5 mg/head/day, Sigma Co.) was dissolved in 70% ethanol and added to the corn-molasses mixture for treatments 3 and 4. A similar volume of ethanol alone was added to the corn-molasses mixture for treatments 1 and 2. Corn was top dressed on pellets at feeding. The corn mixture was stored at 4C and mixed weekly. Rams were fed separately to avoid the effects that melatonin may have on them. Rams were fed .45 kg rolled corn in addition to the pellets. A trace mineralized salt and dicalcium phosphate mixture (50:50) was available free-choice. Sheep were fed at 1300 h in an attempt to keep serum levels of melatonin high for 16 consecutive h in groups 3 and 4. Sheep were weighed every 28 d to monitor weight changes.

Treatments 2 and 4 were kept in a temperature and light controlled room in separate pens. Lights came on at 1100 h. Temperature was maintained at 21C$\pm$1C by heating and cooling systems within the building. The room allowed for 1.5 m^2 per animal. The floor consisted of two 2.43 m wide slotted areas with a 1.52 m wide center concrete alley. Water was supplied via automatic bowl fountains. Floors were scraped daily and the pit flushed three times per wk. Artificial light source in the room was comparable to that in Trial 1.

Treatments 1 and 3 were kept outside without shelter in separate pens providing 44.6 m^2 per animal.

Water was provided by an automatic bowl fountain. The pen area was dirt. Shade was provided by metal roofing.

Blood samples were obtained from these ewes in the same manner as in Trial 1. Blood samples from ewes in treatment 1 and 3 were obtained at 0700 h and from ewes in treatments 2 and 4 at 1100 h. This was done so that all treatments were bled at approximately the same time after the dark phase. Aliquots for prolactin assay were not removed from serum samples at this time.

The experiment terminated on August 24, 1984. Subsequent to this, ewes were exposed to a Columbia clean-up ram. At lambing, ewe number, date and time of birth, sex of lambs, type of birth, and breed of sire were recorded.

Two ewes from group 3 (ND+mel) aborted Suffolk sired lambs. One ewe from group 3 (ND+mel) died prior to lambing. An autopsy report from the South Dakota Veterinary Diagnostic Laboratory indicated that the loss was attributable to ovine progressive pneumonia and other unknown factors, and were not associated with experimental treatment. These 3 ewes were not included in the analysis.

Prolactin and progesterone assays were performed similarly to those in Trial 1.

Melatonin assays were performed on weekly bleedings on a representative portion of the ewes. Blood samples

from five ewes in each treatment were chosen at random for the assay.

Melatonin was measured by radioimmunoassay with Dextran-coated charcoal termination. Serum (300 ul) was combined with 100 ul ^{3}H-melatonin and then allowed to incubate for at least 30 min. After incubation, 2 ml chloroform was added, the mixture vortexed for 15 sec and then shaken for 20 min. After shaking, the aqueous layer was aspirated off and 500 ul of phosphate buffer (pH=7.4) was added. This mixture was then vortexed for 30 sec. Again, the aqueous layer was aspirated off. The remaining chloroform in the tube was then drawn off and put into a 12x75 mm test tube to be dried under nitrogen gas. Two milliliters chloroform was added to the extraction tubes and the process was repeated.

After drying, 400 ul phosphate-gel buffer (pH=7.4) was added to the 12x75 mm tubes and was vortexed for 15 sec. One hundred microliters of this solution was pipetted off and transferred to scintillation vials. Scintillation fluid was added and these were counted for two min in a liquid scintillation counter.

Two milliliters petroleum ether was then added to the tubes. This was vortexed for 25 sec and shaken for 10 min. The petroleum ether was aspirated off and the extract was assayed for melatonin.

Melatonin was labelled with sodium [^{125}I] iodide with specific activity of greater than 2800 uCi/ug by Meloy Laboratories (Springfield, Virginia). ^{125}I-melatonin was diluted to 40,000 cpm/100 ul before use. Maximum binding was 30-35%. Termination of competition was accomplished by adding 100 ul Dextran-coated charcoal, and centrifuging at 20,000 rpm for 20 min. Three hundred microliters was immediately pipetted off and counted in a gamma counter for one min.

The antibody used was diluted to 1:12,000 in phosphate buffer. Sensitivity of the assay was determined to be 5.0 ng/ml. The standard curve was linear between 5.0 ng/ml and 2000 ng/ml. Increasing volumes of wether serum was found to be parallel to the standard curve. Wether serum spiked with melatonin was assayed for recovery. Recovery was 85-95%. Assay values were calculated using a log-logit method. Pooled wether serum and spiked wether serum were used for intra- and interassay coefficients. Coefficients of variation for these were .11 and .16, respectively.

Analysis of the data was similar to that for Trial 1.

RESULTS AND DISCUSSION

Trial 1 - Finnsheep x Targhee Ewes in Treatments 1-5

Starting ewe weights were similiar (P>.05) among treatments (table 1). However, mean ewe weights at the end of the study differed (P<.05). Ewes in treatment 4 (8L:16D+ergo) were lighter at the end of the trial than were ewes in treatments 1 (ND), 2 (8L:16D), or 5 (16L:8D-8L:16D). Average weight gain for ewes in treatment 4 were also less (P<.05) than for ewes in treatments 1,2, or 5 (5.0 kg vs 15.5 kg, 10.3 kg and 9.5 kg, respectively). Ewes in treatment groups receiving ergo averaged less weight gain, possibly due to the stress associated with twice weekly injections.

There were no differences in the number of breeding marks or in the days to the first or second breeding mark for any treatment (P>.05). However, days to first mark had a large numerical range (table 2). Ewes in natural daylight treatments averaged fewer days to first mark than did artificial lighting treatments. Days to second mark did not show this trend, and ranged from 52 to 76 d.

Table 3 shows mean lambing and conception dates, and reaction period (mean days from start of the experiment to conception). Nine ewes conceived within 20 d of the experiment start. All of these ewes were in treatment 1 or 3 (ND or ND+ergo). This accounts for the early average lambing date in these treatments for the 50% that did lamb.

TABLE 1. LEAST SQUARES MEANS AND STANDARD ERRORS FOR BEGINNING WEIGHT, ENDING WEIGHT AND WEIGHT CHANGE FOR FINNSHEEP X TARGHEE EWES IN TRIAL 1

Treatment	No. ewes	Beginning wt, kg	Ending wt, kg	Wt change, kg
ND	10	68.1±3.16	83.6±3.38[a]	15.5±2.19[a]
8L:16D	10	73.6±3.16	83.9±3.38[a]	10.3±2.19[ab]
ND+ergo	10	67.8±3.16	76.8±3.38[ab]	9.0±2.19[ab]
8L:16D+ergo	10	65.5±3.16	70.5±3.38[b]	5.0±2.19[b]
16L:8D-8L:16D	10	73.7±3.16	83.2±3.38[a]	9.5±2.19[ab]

[a,b] Numbers in the same column with different superscripts differ ($P<.05$).

TABLE 2. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF BREEDING MARKS AND DAYS TO FIRST MARK FOR FINNSHEEP X TARGHEE EWES (TRIAL 1)

Treatment	No. ewes receiving marks	No. marks	Days to first mark
ND	8	1.6±.37	32±9.1
8L:16D	9	1.9±.37	42±8.6
ND+ergo	9	1.8±.37	17±9.1
8L:16D+ergo	9	2.1±.37	41±8.6
16L:8D-8L:16D	8	1.4±.37	55±9.1

This is also consistant with the earlier days to first mark seen for these ewes. Average conception dates were May 29 and June 14 for ewes in the ND and ND+ergo treated ewes, respectively. These differed ($P<.05$) from all inside treatments. Why early conception occurred in the ewes treated with natural daylight only is not known. It would appear that the ewes that conceived very early in the study were not anestrus at the time the trial was initiated. Progesterone values for some of these ewes were higher than expected, indicating that some ewes were cycling. A reaction period of only 12 d (less then one cycle) seen for the ewes in the ND treatment supports this.

TABLE 3. LEAST SQUARES MEANS FOR CONCEPTION DATE, LAMBING DATE AND REACTION PERIOD FOR FINNSHEEP X TARGHEE EWES (TRIAL 1)

Treatment	No. ewes	Conception date	Lambing date	Reaction period, d
ND	10	5-28-83 [b]	10-25-83 [b]	12 [b]
8L:16D	10	7- 9-83 [a]	12- 2-83 [a]	54 [a]
ND+ergo	10	6-14-83 [b]	11- 7-83 [b]	29 [b]
8L:16D+ergo	10	7-13-83 [a]	12- 6-83 [a]	58 [a]
16L:8D-8L:16D	10	7-29-83 [a]	12-22-83 [a]	74 [a]

[a,b] Numbers in the same column with different superscripts differ ($P<.05$).

The reaction period for ewes in the 8L:16D treatments indicate that an average of 55 d is needed to initiate fertile cycles in these ewes. As would be expected, ewes in treatment 5 (16L:8D-8L:16D) had the longest reaction interval. Hart (1950) supports this finding that a slow decrease in light is not necessary to initiate cyclic activity in anestrous ewes.

Table 4 presents the lambing data on these ewes. Ninety-five percent of the ewes in artificial lighting lambed compared to 50% of the ewes in natural daylight treatments ($P<.05$). No differences were seen in the treatments under artificial lighting or in the treatments in natural daylight. Number of lambs born per ewe lambing was greatest for the ewes in the 8L:16D and the 8L:16D+ergo treatments (2.6 lambs per ewe lambing). This differed ($P<.05$) from ewes in the ND treatment (1.6 lambs per ewe lambing). Average litter weight of lambs born per ewe lambing was 11.2 kg for ewes in treatment 2 (8L:16D), but only 7.3 kg for ewes in treatment 1 (ND, $P<.05$). All other treatments did not differ. This difference may be due to heat stress in the ewes in natural daylight. Yeates (1956) reported that high temperatures decrease the number of lambs born and the birth weight of lambs born per ewe lambing. This phenomenon is reported to be especially severe in the early stages of pregnancy.

TABLE 4. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF LAMBS AND WEIGHT OF LAMBS PER EWE LAMBING FOR FINNSHEEP X TARGHEE EWES (TRIAL 1)

Treatment	No. ewes	No. ewes lambing	No. lambs/ ewe lambing	Litter wt/ ewe lambing, kg
ND	10	5[a]	1.6±.32[b]	7.3±1.16[b]
8L:16D	10	8[ab]	2.6±.25[a]	11.2±0.91[a]
ND+ergo	10	5[a]	2.0±.32[ab]	9.1±1.20[ab]
8L:16D+ergo	10	10[b]	2.6±.23[a]	9.6±0.82[ab]
16L:8D 8L:16D	10	10[b]	2.2±.23[ab]	10.3±0.82[ab]

a,b Numbers in the same column with different superscripts differ (P<.05).

Hackett and Wolynetz (1985) suggests that fertility is increased when lighting is changed abruptly rather than slowly. This trial may show a trend in this direction as ewes in 16L:8D-8L:16D lambed an average of 2.2 lambs per ewe lambing, while ewes in the 8L:16D treatments lambed an average of 2.6 lambs per ewe lambing.

Figure 2 to 6 depict serum prolactin and progesterone values for these ewes at weekly intervals.

Serum prolactin values showed overall treatment differences with ewes in the ND treatment having a mean of 246 ng/ml. This was higher (P<.05) than ewes in all other

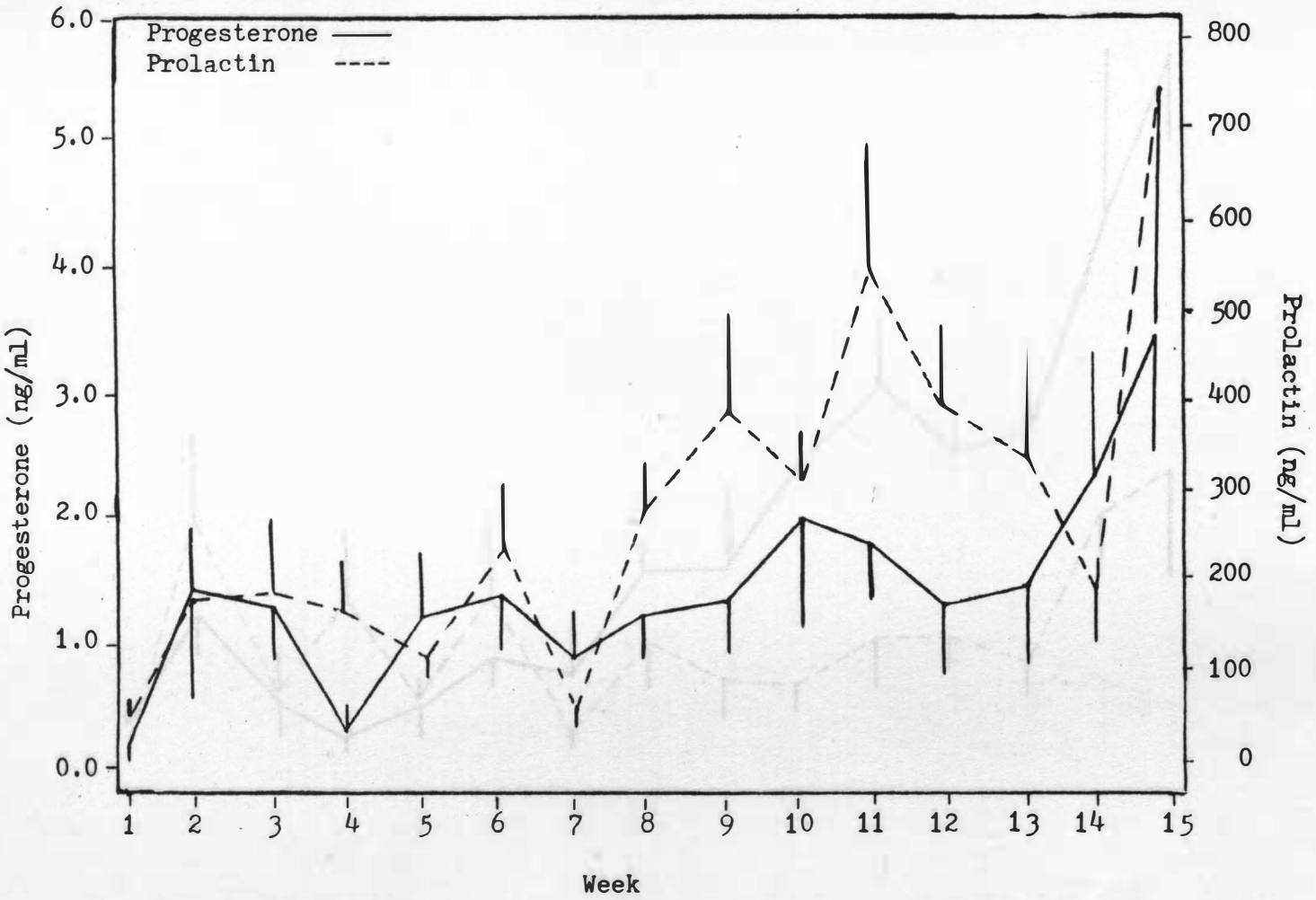


Figure 2. Prolactin and progesterone levels by week for Trial 1, FT ewes in treatment 1 (ND).

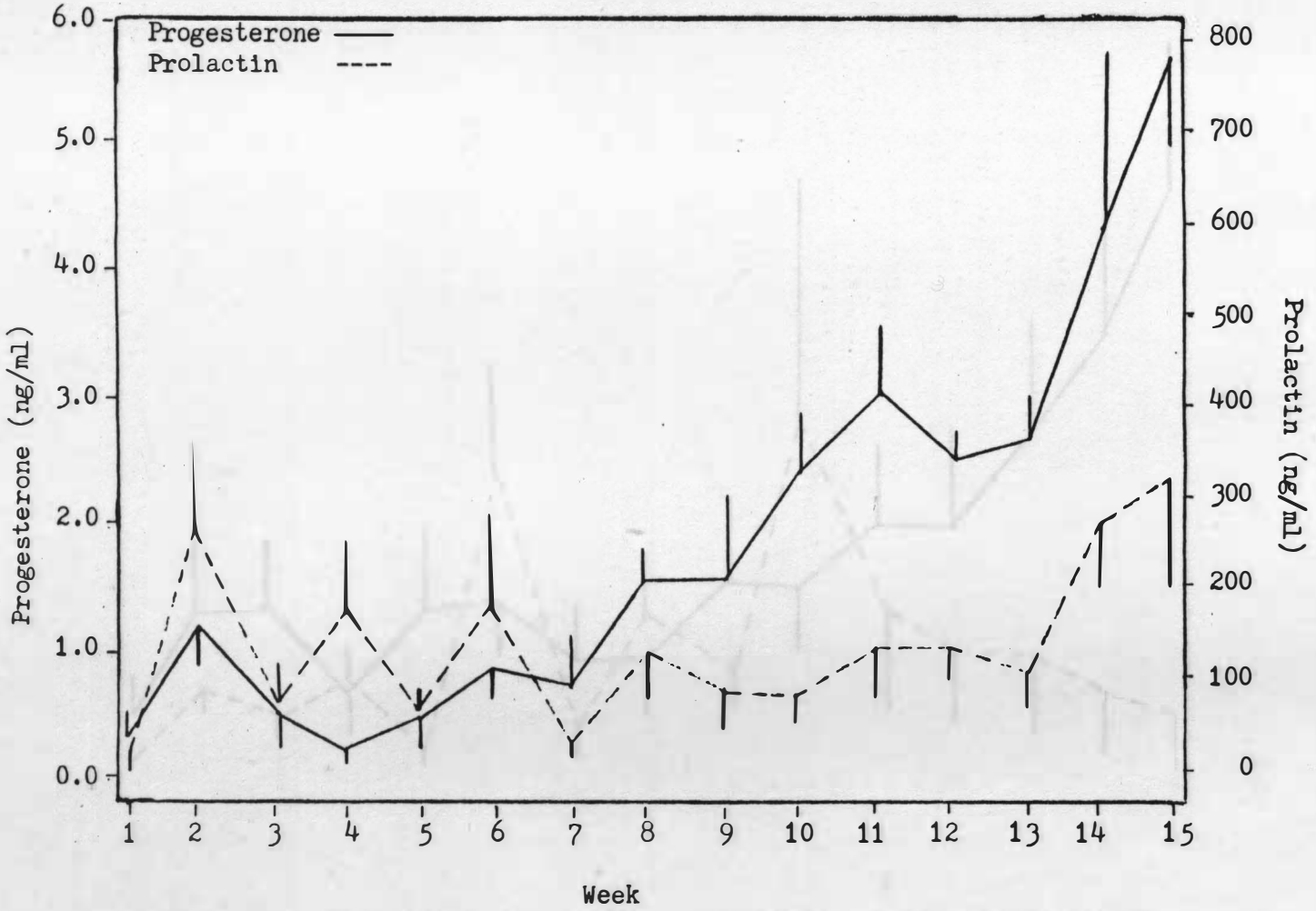


Figure 3. Prolactin and progesterone levels by week for Trial 1, FT ewes in treatment 2 (8L:16D).

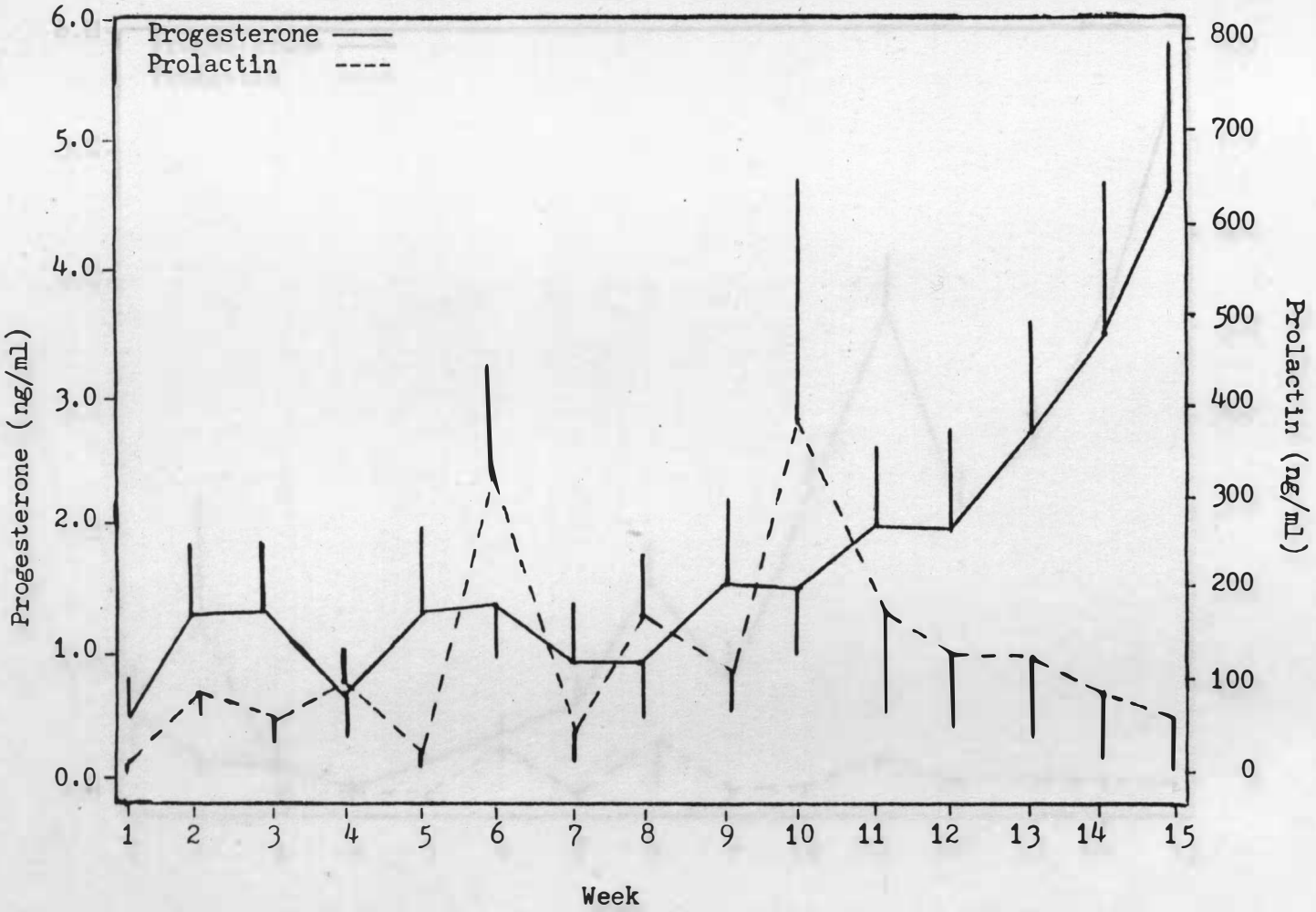


Figure 4. Prolactin and progesterone levels by week for Trial 1, FT ewes in treatment 3 (ND+ergo).

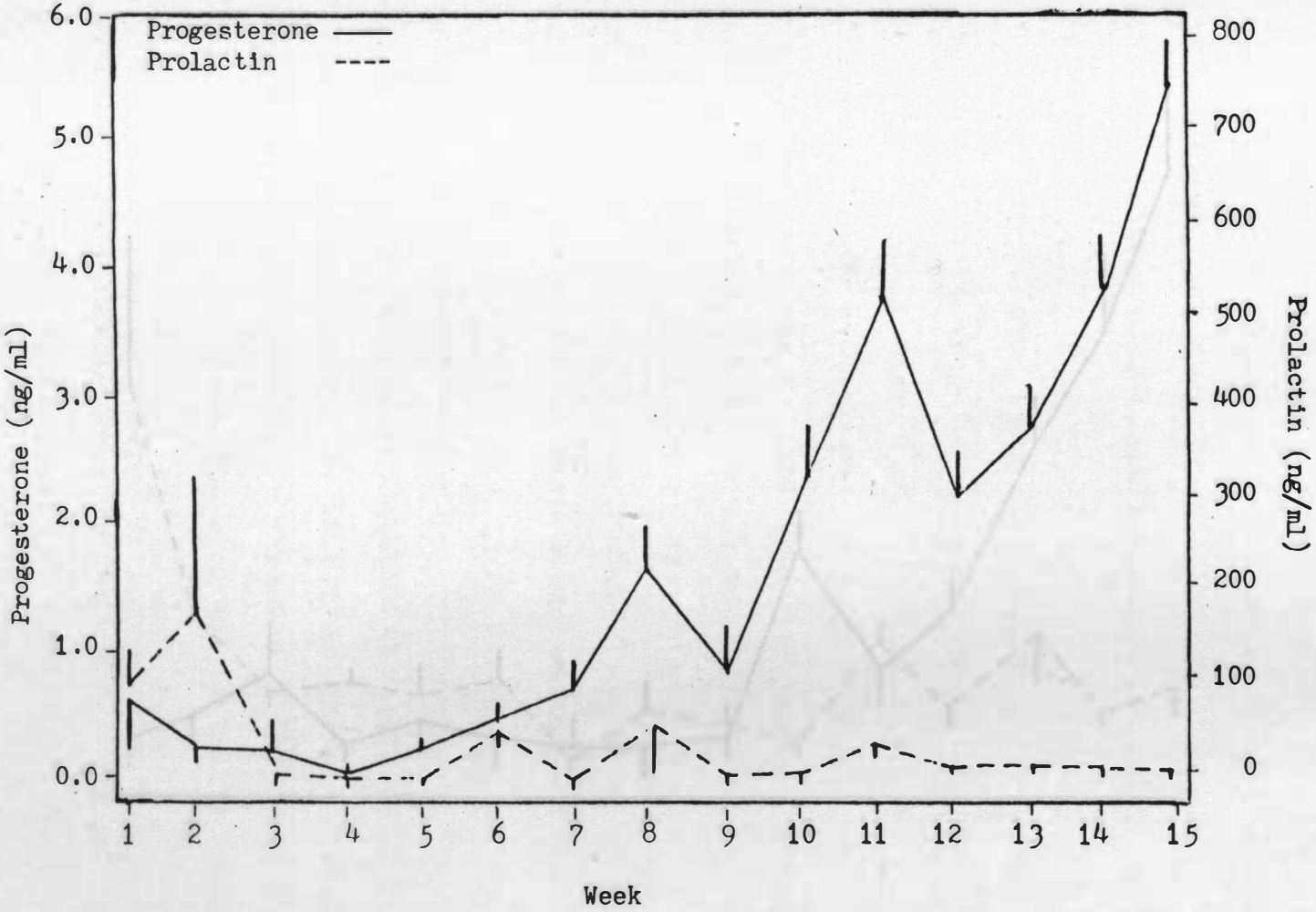


Figure 5. Prolactin and progesterone levels by week for Trial 1, FT ewes in treatment 4 (8L:16D+ergo).

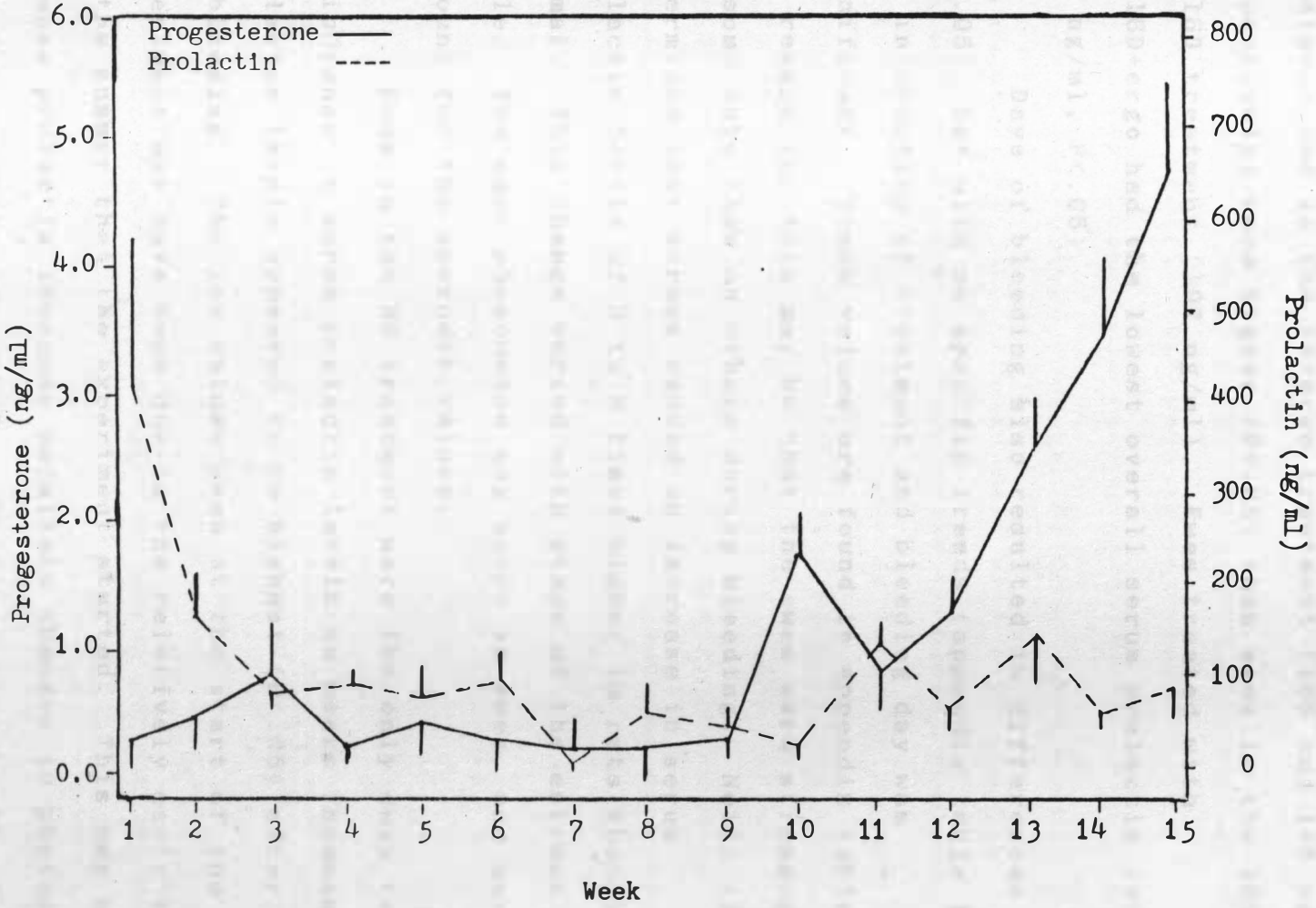


Figure 6. Prolactin and progesterone levels by week for Trial 1, FT ewes in treatment 5 (16L:8D-8L:16D).

treatments. Serum prolactin levels for ewes in the 8L:16D treatment and in the ND+ergo treatment (166 and 145 ng/ml, respectively) were higher ($P<.05$) than ewes in the 16L:8D-8L:16D treatment (107 ng/ml). Ewes treated with 8L:16D+ergo had the lowest overall serum prolactin levels (27 ng/ml, $P<.05$).

Days of bleeding also resulted in differences ($P<.05$), but with no specific trends (appendix table 1). The interaction of treatment and bleeding day was significant. These values are found in appendix table 2. The reason for this may be that the ewes were stressed more on some days than on others during bleeding. Neill (1970) determined that stress caused an increase in serum prolactin levels of 3 to 8 times higher in rats than is normal. This change varied with stage of the estrous cycle. The same phenomenon may occur in ewes, and may account for the sporadic values.

Ewes in the ND treatment were the only ewes to show an increase in serum prolactin levels as weeks increased. Prolactin levels appeared to be highest ($P<.05$) after wk 11 of bleeding. The low values seen at the start of the experiment may have been due to the relatively early time of the summer that the experiment started. This may be because prolactin increase parallels changes in photoperiod (Webster and Haresign, 1983). This may also be the reason that ewes in the 8L:16D treatment appeared to have a fairly

consistant level of serum prolactin throughout the experiment.

The ewes showing the most rapid and consistant drop in serum prolactin level were those treated with 8L:16D+ergo. These ewes showed no differences in serum prolactin levels across weeks ($P>.05$). This agrees with Niswender (1974) who treated ewes with ergo and saw a decrease in serum prolactin levels to almost 0 ng/ml. Ewes treated with ND+ergo did not show as large or as sustained a drop in serum prolactin, but prolactin levels were decreased to below control ewes (ND). This may have been because the ewes in ND and receiving ergo had a more substantial prolactin release to halt because of a longer light cycle and because of the stress associated with environmental conditions (heat, etc.).

Ewes treated with 16L:8D-8L:16D also had a very rapid decline in serum prolactin level. The reason for this is unknown. It would be expected that prolactin release would decline slowly, following the photoperiod. The nadir of the cycle would be expected to be approximately wk 8 when the light period was shortest.

Treatment also resulted in serum progesterone differences (table 5). Ewes in the 8L:16D, 8L:16D+ergo and ND+ergo treatments had the highest overall mean values, indicating that ewes in these treatments may have cycled

more, conceived sooner, or had more corpora lutea than other treatments (Thornburn et al., 1969 and Basset et al., 1969). Ewes in the ND treatment did not differ ($P>.05$) from those ewes receiving ergo, but did have lower mean serum progesterone levels than those ewes in the 8L:16D treatment. Ewes in the 16L:8D-8L:16D treatment did not differ ($P>.05$) from ewes in natural daylight but did have lower average progesterone values than did ewes in all other treatments ($P<.05$).

Weeks resulted in differences ($P<.05$) in serum progesterone levels (appendix table 3). Later weeks had higher progesterone levels than did earlier weeks. This is expected because more ewes started to cycle and conceive as the experiment progressed, and so serum progesterone was elevated in more ewes. The interaction of treatment by week for serum progesterone level was not significant ($P>.05$).

Ewes in the 8L:16D and the 8L:16D+ergo treatments had the earliest significant increase in serum progesterone level, starting at wk 11 and 10, respectively ($P<.05$). Ewes in the ND, ND+ergo, and 16L:8D-8L:16D treatments did not show this substantial of an increase until wk 15, 14, and 13, respectively ($P<.05$).

Correlations of progesterone and prolactin by week showed no significance ($P>.05$).

TABLE 5. LEAST SQUARES MEANS AND STANDARD ERRORS FOR SERUM PROGESTERONE LEVEL

Treatment	Progesterone, ng/ml
ND	1.5±.12 bc
8L:16D	1.9±.12 a
ND+ergo	1.8±.12 ab
8L:16D+ergo	1.7±.12 ab
16L:8D-8L:16D	1.2±.12 c

a,b,c Numbers differ at P<.05.

It appears that reduced light will hasten cyclic activity and increase conception in the normally anestrous ewe. Ergocryptine appears to have no effect on initiating cyclic activity in anestrous ewes, although it will substantially decrease serum prolactin levels. A gradual increase in the length of the dark phase seems to have no additional benefits.

Trial 1-Finnsheep x Targhee and Targhee Ewes in Treatments 1 and 2

Finnsheep x Targhee (FT) crossbred ewes and straightbred Targhee (T) ewes were used to compare breed differences in treatments 1 (ND) and 2 (8L:16D). Initial weights varied ($P<.05$), probably due to breed differences. Final weights and weight changes were similar for all treatments ($P>.05$, table 6).

Breeding mark data is shown in table 7. No treatment or breed differences were observed ($P>.05$).

FT ewes in the ND treatment conceived and lambed an average of 39 d earlier ($P<.05$) than ewes of either breed in the 8L:16D treatment group (table 8). Possible reasons for this were discussed earlier. It appears that no breed

TABLE 6. LEAST SQUARES MEANS AND STANDARD ERRORS FOR BEGINNING WEIGHT, ENDING WEIGHT AND WEIGHT CHANGE FOR T AND FT EWES IN TREATMENTS 1 AND 2

	ND		8L:16D	
Item	T	FT	T	FT
Start wt., kg	61.4±2.6[a]	68.1±2.6[ab]	66.5±2.6[ab]	73.6±2.6[b]
End wt., kg	78.7±2.7	83.6±2.7	76.6±2.7	83.9±2.7
Wt. change, kg	17.3±2.4	15.5±2.4	10.1±2.4	10.3±2.4

[a,b] Numbers in the same row with different superscripts differ ($P<.05$).

TABLE 7. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF BREEDING MARKS AND DAYS TO FIRST MARK FOR FT AND T EWES IN TREATMENTS 1 AND 2

	ND		8L:16D	
Item	T	FT	T	FT
No. ewes marked	6	8	8	9
No. marks	1.0±.35	1.6±.35	1.3±.35	1.9±.35
Days to mark 1	55±11.3	32±11.3	37±13.1	42±10.7

TABLE 8. LEAST SQUARES MEANS FOR CONCEPTION DATE, LAMBING DATE AND REACTION PERIOD FOR T AND FT EWES IN TREATMENTS 1 AND 2

	ND		8L:16D	
Item	T	FT	T	FT
No. ewes	10	10	10	10
No. lambing	6	5	10	8
Conception date	6-25-83 [ab]	5-28-83 [a]	7-10-83 [b]	7-9-83 [b]
Lambing date	11-18-83 [ab]	10-25-83 [a]	12- 3-83 [b]	12- 2-83 [b]
Reaction period, d	40 [ab]	12 [a]	55 [b]	54 [b]

[a,b] Numbers in the same row with different superscripts differ ($P<.05$).

differences occurred, especially in the 8L:16D treatment. The 8L:16D treatment showed only one d difference ($P>.05$) in conception date, lambing date, and reaction period for the two different breeds. Ewes in the ND treatment indicated a larger numerical difference, but still no statistical difference ($P>.05$).

Table 9 shows that the number of ewes lambing in each treatment and breed group did not differ ($P>.05$). However, pooled ND treatment data indicated fewer ewes lambing ($P<.10$) than did pooled values for the ewes in the

TABLE 9. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF LAMBS BORN AND LITTER WEIGHT OF LAMBS BORN PER EWE LAMBING FOR T AND FT EWES IN TREATMENTS 1 AND 2

	ND		8L:16D	
Item	T	FT	T	FT
No. ewes	10	10	10	10
No. ewes lambing	6	5	10	8
No. lambs per ewe lambing	$1.0^{a}\pm.19$	$1.6^{b}\pm.21$	$1.6^{b}\pm.15$	$2.6^{c}\pm.17$
Litter wt., kg	$6.2^{a}\pm.95$	$7.3^{ab}\pm.95$	$9.0^{b}\pm.67$	$11.2^{c}\pm.75$

a,b,c Numbers in the same row with different superscripts differ ($P<.05$).

8L:16D treatment. In the ND and 8L:16D treatments, the FT ewes averaged .6 and 1.0 more lambs per ewe lambing, respectively, than did T ewes ($P<.05$). This is in agreement with Donald and Read (1967) who found that a Finnsheep flock with no yearlings averaged 2.53 lambs per ewe exposed.

Litter weight of lambs born per ewe lambing did not differ for lambs from the two breeds in the ND treatment ($P>.05$). However, FT in the 8L:16D treatment had 2.2 kg more lamb per ewe lambing than did T ewes in that treatment ($P<.05$). Other than differences caused by the FT's natural ability to conceive and carry more lambs to birth, there appears to be no breed differences between the two when using lighting regimes to induce ewes to cycle in the normal summer anestrous season.

Figures 7 and 8 depict serum prolactin and progesterone levels for the two groups at weekly intervals. Serum prolactin values showed overall treatment differences ($P<.05$) with the two breeds combined. Ewes in the ND treatment averaged a serum prolactin level of 266 ng/ml while ewes in the 8L:16D treatment averaged serum prolactin levels of 135 ng/ml. This is in agreement with Webster and Haresign (1983) who stated that changes in prolactin release parallel changes in photoperiod. Day of bleeding also differed ($P<.05$) but with no specific trends (appendix table 4). These differences may be due to photoperiod or

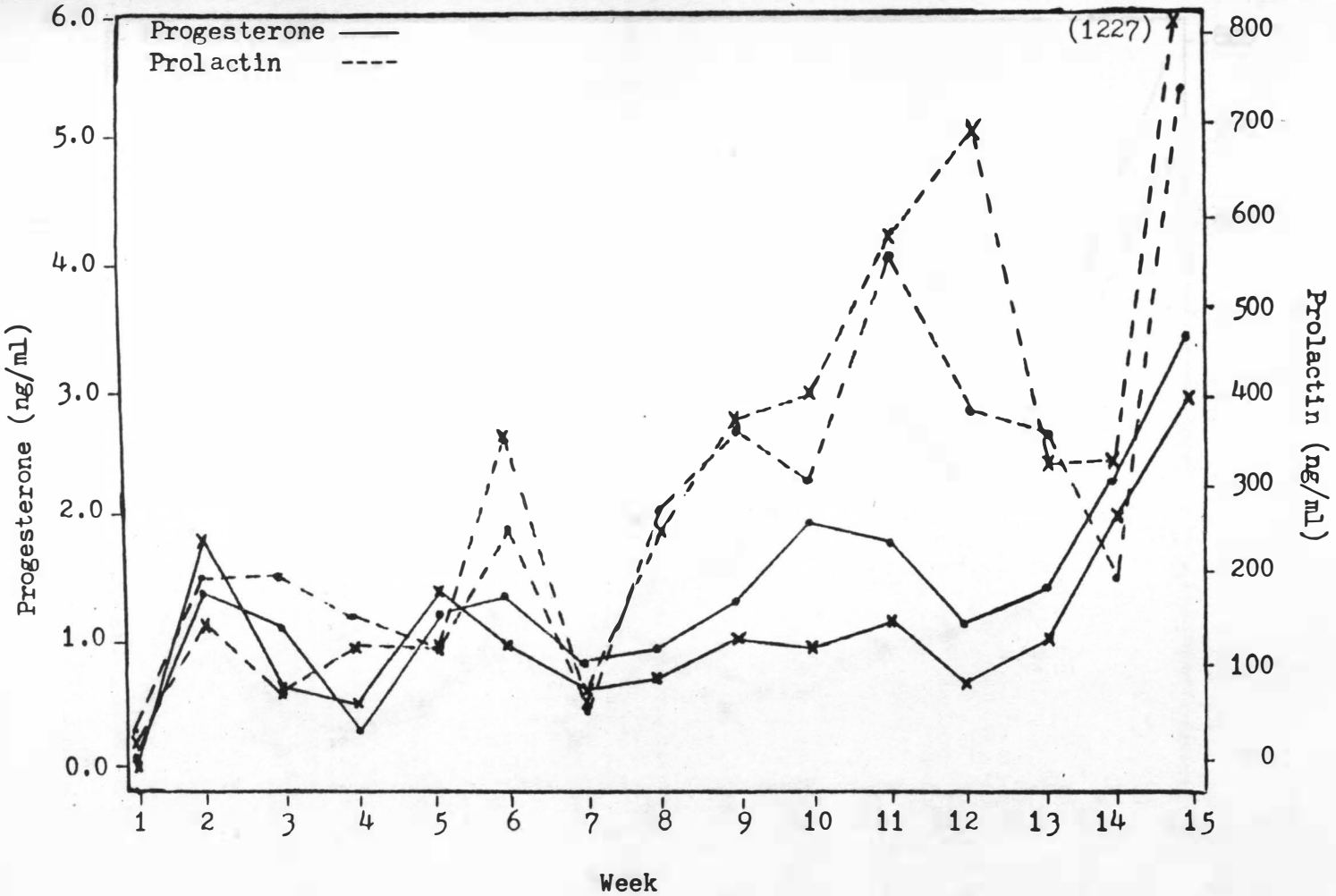


Figure 7. Prolactin (S.E.~96.7) and progesterone (S.E.~.44) levels by week for Trial 1, T and FT ewes in treatment 1 (T = x, FT = ·).

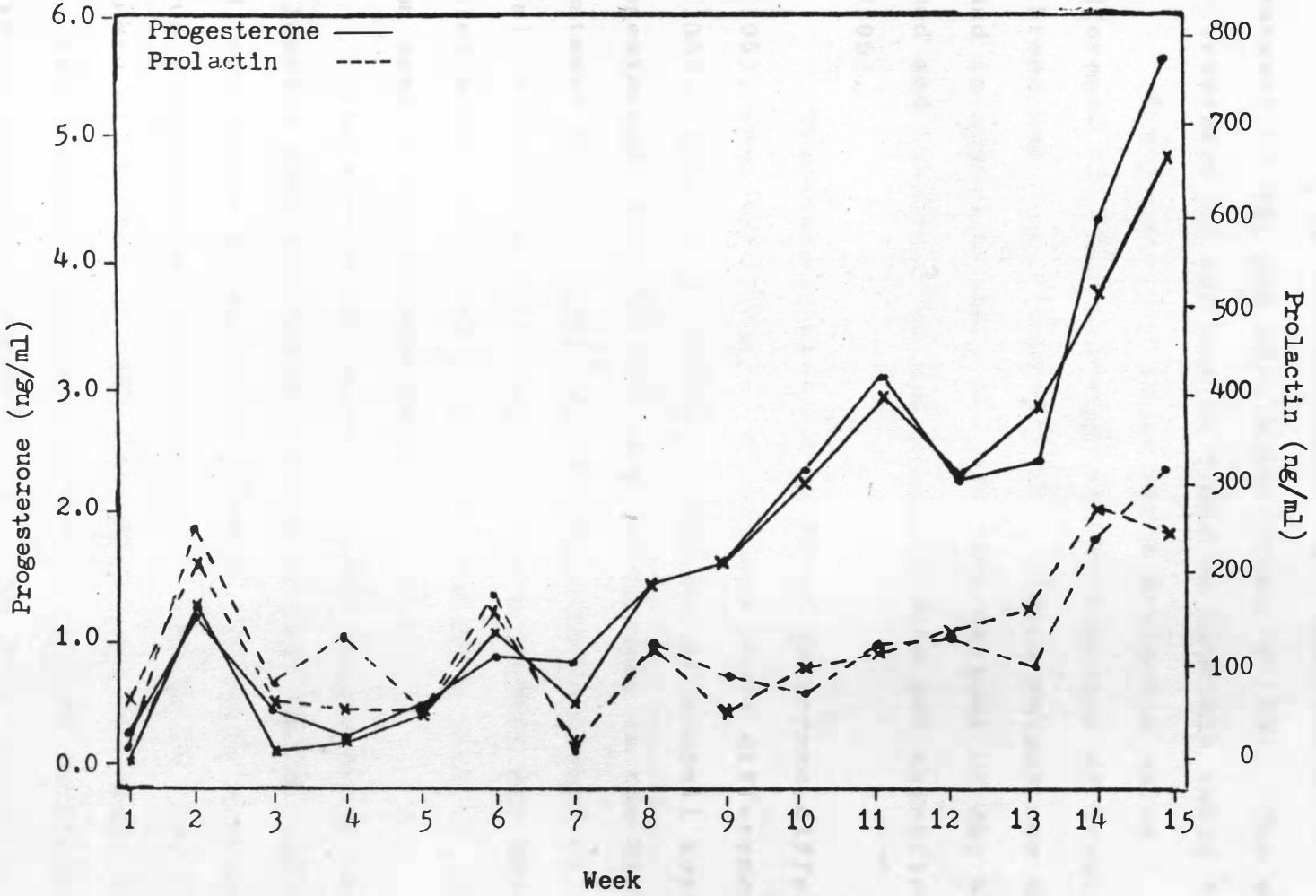


Figure 8. Prolactin (S.E.~96.7) and progesterone (S.E.~.44) levels by week for Trial 1, T and FT ewes in treatment 2 (T = x, FT = ·).

to the stress factors involved. The interaction of treatment by day was also significant ($P<.05$). The means for treatment by day can be found in appendix table 5.

Breed resulted in no serum prolactin value difference ($P>.05$), although the interaction of treatment by breed was significant ($P<.05$). These values can be found in appendix table 6. The interactions of day by breed and treatment by day by breed were not significant ($P<.05$).

Treatment indicated serum progesterone differences ($P<.05$), week differences ($P<.05$) and breed differences ($P<.05$). Ewes in the ND treatment had an overall serum progesterone level of 1.3 ng/ml while ewes in the 8L:16D treatment had an overall serum progesterone level of 1.8 ng/ml, indicating that ewes in this treatment may have cycled more, conceived sooner, or had more corpora lutea than ewes in the ND treatment.

The week differences in serum progesterone level followed a specific trend. Weeks earlier in the experiment had lower serum progesterone values. As weeks increased, serum progesterone values increased. Weeks 11, 13, 14 and 15 were all higher ($P<.05$) than wk 1 to 9 (appendix table 7). This would be expected because, as more ewes start to cycle and conceive, serum progesterone level would be elevated in these ewes.

The interaction of treatment by week was significant ($P<.05$). These values are found in appendix table 8. The interaction of treatment by week by breed was also significant ($P<.05$). These values are found in figures 7 and 8.

The breed difference ($P<.05$) shown in overall serum progesterone level was 1.4 ng/ml for T ewes and 1.7 ng/ml for FT ewes. This breed difference would be expected since FT ewes had more lambs, and so probably ovulated more and had more corpora lutea. Basset et al. (1969) discovered that an increased number of corpora lutea will increase serum progesterone levels.

Correlations between progesterone and prolactin between breeds and within breeds by week were not significant ($P>.05$).

The only breed differences in hormone values between T and FT ewes appears to be due to the FT ewe's natural ability to ovulate more ova, and therefore form more corpora lutea than the T ewes.

Trial 2

As table 10 indicates, ewe starting weight, ending weight, and weight change throughout this trial were similar between treatments (P>.05).

The number of breeding marks per ewe and days to first mark did not differ between treatments (P>.05, table 11).

TABLE 10. LEAST SQUARES MEANS AND STANDARD ERRORS FOR BEGINNING WEIGHT, ENDING WEIGHT AND WEIGHT CHANGE FOR TRIAL 2

Treatment	No. ewes	Beginning weight, kg	Ending weight, kg	Weight change, kg
ND	15	70.0±2.31	75.1±3.01	5.1±1.73
8L:16D	15	70.1±2.32	77.7±2.40	7.6±1.22
ND+mel	12	71.8±2.22	78.4±2.54	6.6± .76
8L:16D+mel	15	65.3±2.65	72.3±2.42	7.0±1.27

TABLE 11. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF BREEDING MARKS AND DAYS TO FIRST MARK FOR EWES IN TRIAL 2

Treatment	No. ewes marked	No. breeding marks	No. days to first mark
ND	15	2.9±.22	12.9±2.76
8L:16D	15	2.5±.31	26.5±5.32
ND+mel	12	3.3±.30	14.3±2.41
8L:16D+mel	15	2.9±.26	17.7±4.21

Table 12 shows the least squares means for conception date, lambing date and reaction period. As shown, the earliest average conception date was July 26 for ewes in the 8L:16D+mel treatment. This was 10 d earlier than the average conception date for ewes in the ND treatment ($P<.05$). An average lambing date of December 21 for ewes in the 8L:16D+mel treatment and of December 31 for ewes in the ND treatment followed this same trend ($P<.05$), as did reaction periods ($P<.05$) of 55 and 65 d, respectively.

It is interesting to note the numerical difference in reaction period, mean conception date, and mean lambing date of 6 to 9 d between those ewes receiving melatonin and those ewes not receiving melatonin.

TABLE 12. LEAST SQUARES MEANS FOR CONCEPTION DATE, LAMBING DATE AND REACTION PERIOD FOR TRIAL 2

Treatment	No. ewes	Conception date	Lambing date	Reaction period, d
ND	15	8- 5-84[a]	12-31-84[a]	65[a]
8L:16D	15	8- 2-84[ab]	12-28-84[ab]	62[ab]
ND+mel	12	7-27-84[ab]	12-22-84[ab]	56[ab]
8L:16D+mel	15	7-26-84[b]	12-21-84[b]	55[b]

[a,b] Numbers in the same column with different superscripts differ ($P<.05$).

Table 13 shows the least squares means and standard errors for the number of lambs born and litter weight of lambs born per ewe lambing, as well as the number of ewes lambing in each treatment. Forty percent of the ewes in the ND treatment lambed (6 out of 15), compared to 95.2% in the other three treatments (15 out of 15 ewes in the 8L:16D treatment, 11 out of 12 ewes in the ND+mel treatment, and 14 out of 15 ewes in the 8L:16D+mel treatment).

The number of lambs born per ewe lambing differed between treatments. Ewes in the 8L:16D and the 8L:16D+mel treatments had 2.3 and 2.4 lambs per ewe lambing, respectively, while ewes in the ND and ND+mel treatments had 1.5 and 1.8 lambs per ewe lambing, respectively ($P<.05$). Litter weight of the lambs born per ewe lambing followed this same trend. Ewes in the 8L:16D and 8L:16D+mel treatments had average litter weights of 10.3 kg while ewes in the ND and ND+mel treatments had average litter weights of 7.2 kg per ewe lambing ($P<.05$). Heat stress on the ewes caused by normal summer temperatures may have contributed to this phenomenon. Yeates (1956) reported that high temperatures decreased birth weight and number of lambs born per ewe lambing.

It appears that reduced light (8L:16D) and(or) melatonin feeding will increase cyclic activity and conception in the normally anestrous ewe. This agrees with

TABLE 13. LEAST SQUARES MEANS AND STANDARD ERRORS FOR NUMBER OF LAMBS BORN AND LITTER WEIGHT OF LAMBS BORN PER EWE LAMBING IN TRIAL 2

Treatment	No. ewes	No. ewes lambing	No. lambs per ewe lambing	Litter wt. per ewe lambing
ND	15	6[a]	1.5±.21[a]	7.3±.90[a]
8L:16D	15	15[b]	2.3±.19[b]	10.5±.69[b]
ND+mel	12	11[b]	1.8±.26[a]	7.1±.68[a]
8L:16D+mel	15	14[b]	2.4±.27[b]	10.1±.72[b]

[a,b] Numbers in the same column with different superscripts differ ($P<.05$).

Nett and Niswender (1982) who indicate that feeding melatonin at a level of 2.5 mg per head per day can hasten the onset of the breeding season in the fall.

Figures 9 to 12 depict serum progesterone and prolactin levels for these ewes across weeks. Serum prolactin levels differed for overall treatment mean ($P<.05$). Ewes in the ND treatment had an overall mean serum prolactin level of 259 ng/ml, which was higher ($P<.05$) than 190 ng/ml for ewes in the 8L:16D treatment, 191 ng/ml for ewes in the ND+mel treatment and 196 for ewes in the 8L:16D+mel treatment. Kennaway and coworkers (1982a) noted that melatonin feeding decreased serum prolactin levels. The same effect is seen in comparing the ewes in the ND treatment with the ewes in the ND+mel

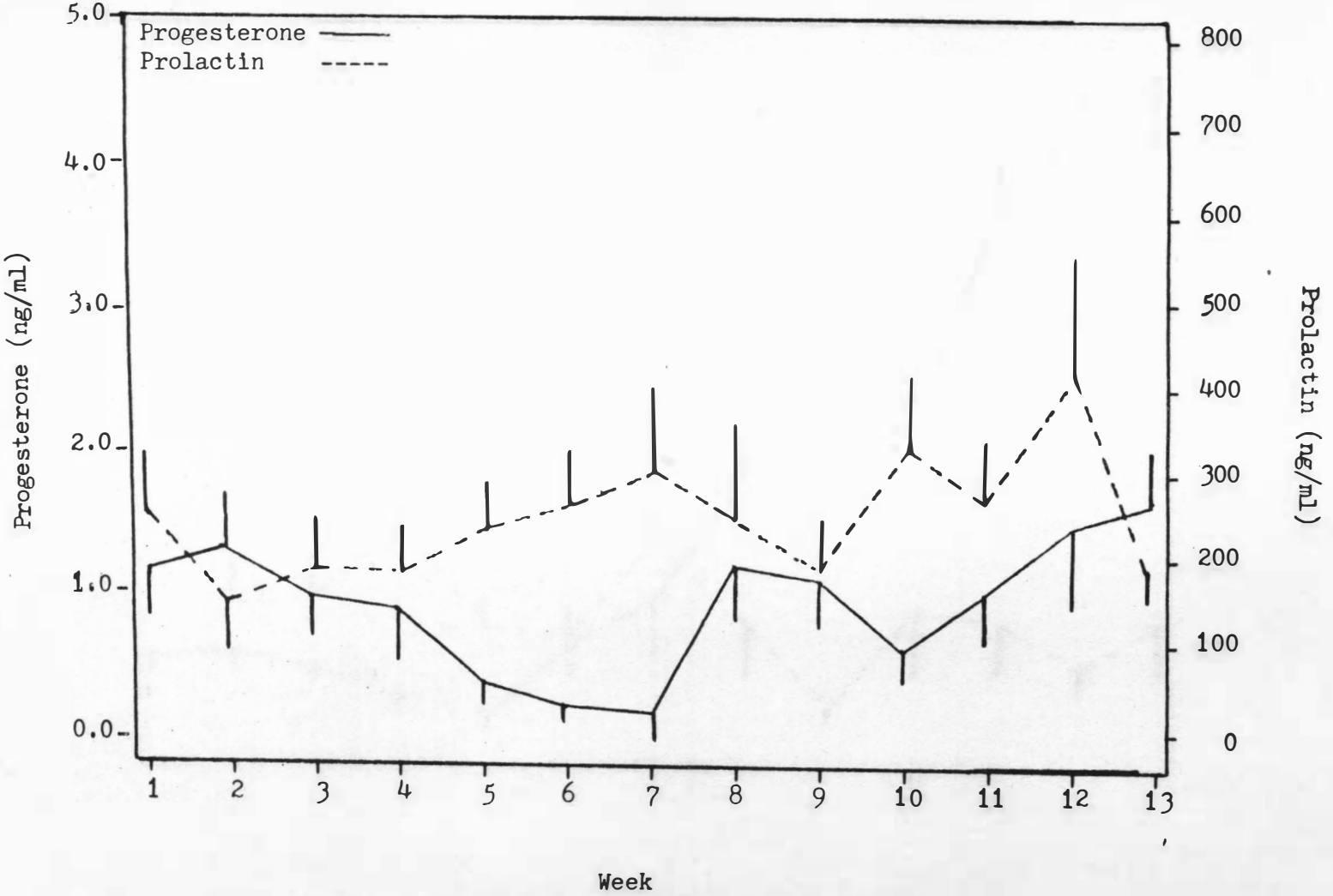


Figure 9. Prolactin and progesterone levels by week for Trial 2, treatment 1 (ND).

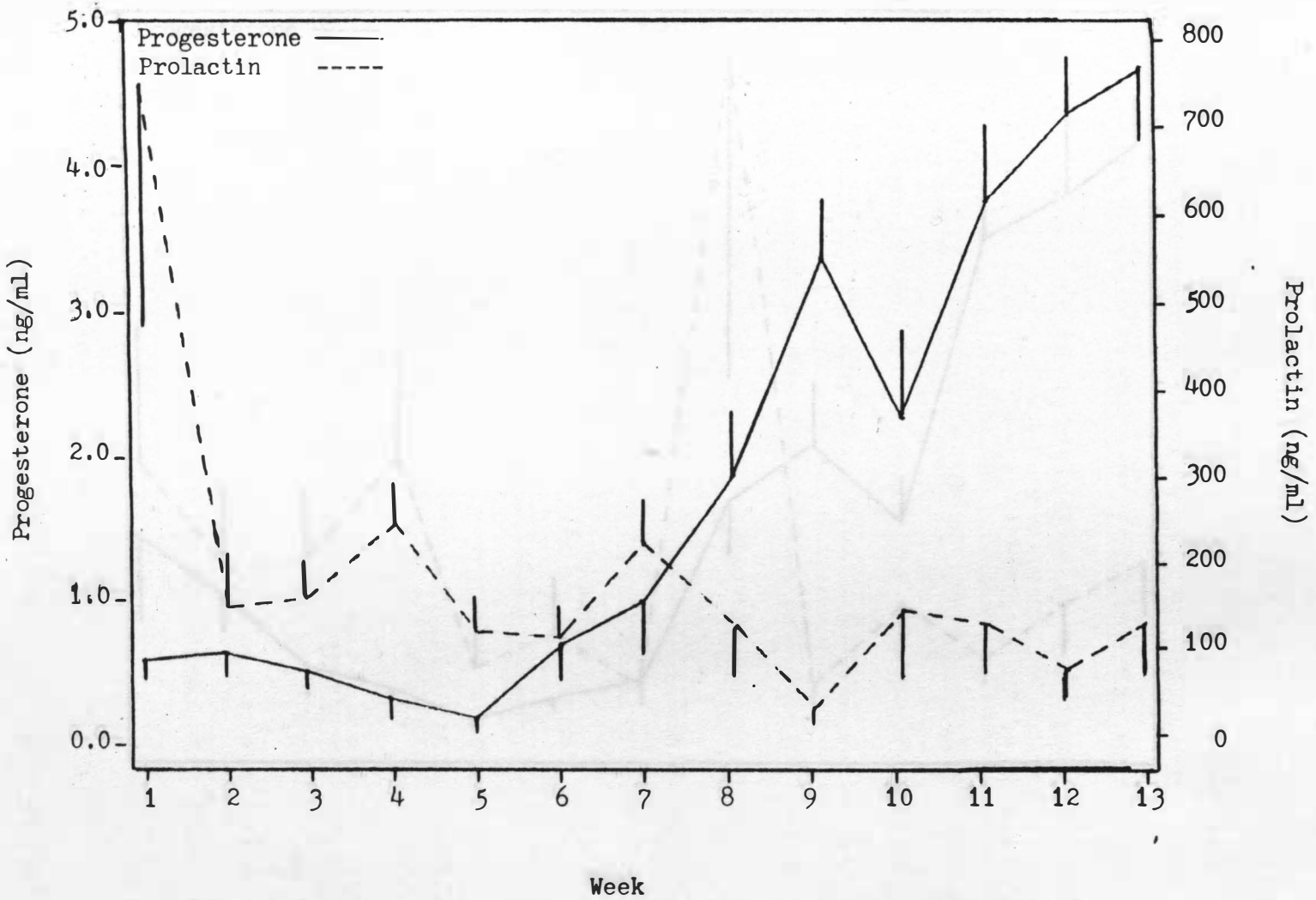


Figure 10. Prolactin and progesterone levels by week for Trial 2, treatment 2 (8L:16D).

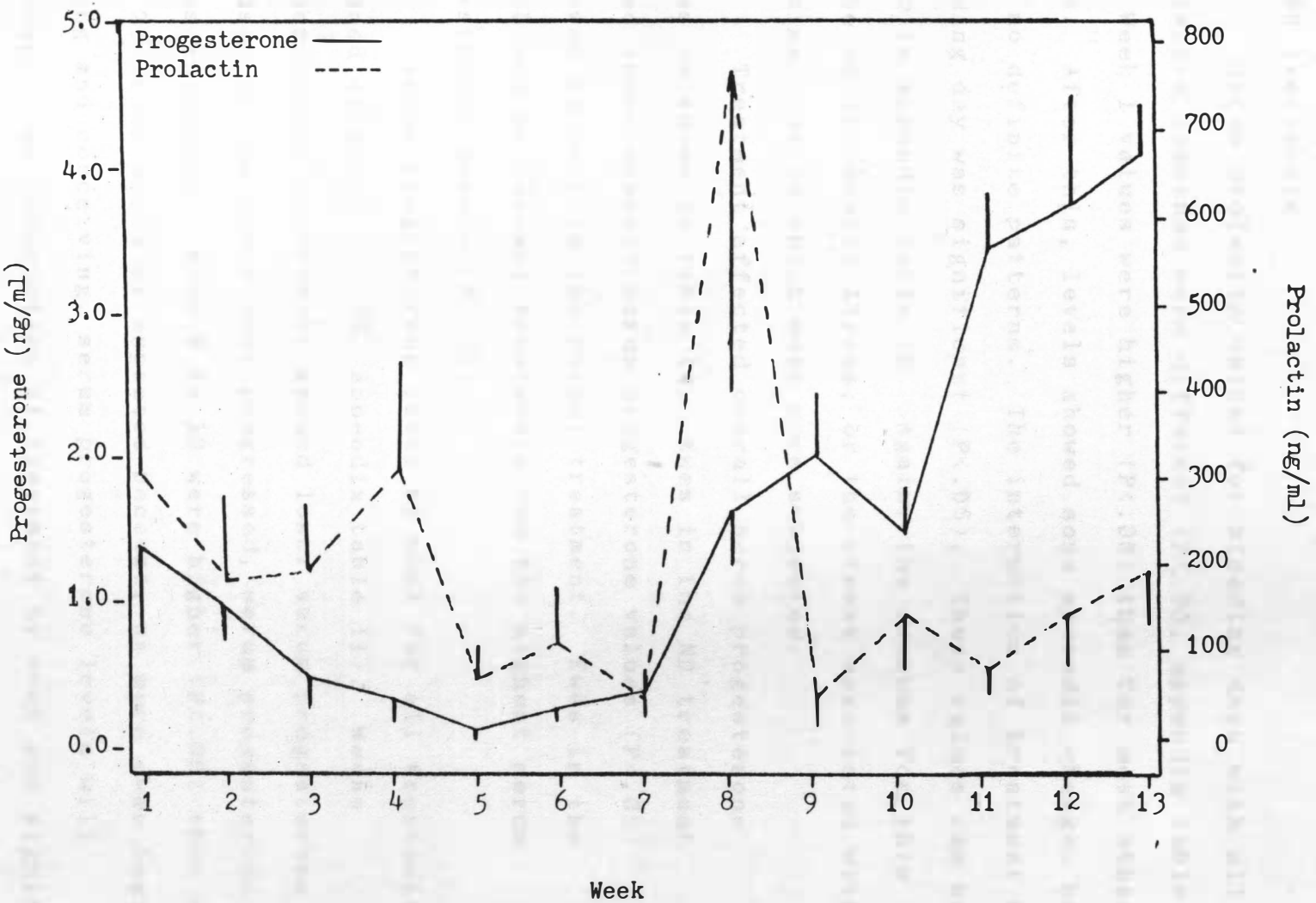


Figure 11. Prolactin and progesterone levels by week for Trial 2, treatment 3 (ND+mel).

treatment, but was not seen with the ewes kept in the 8L:16D treatments.

Serum prolactin values for bleeding days with all treatments combined were different ($P<.05$, appendix table 9). Week 1 values were higher ($P<.05$) than for most other weeks. After this, levels showed some sporadic change, but with no definite patterns. The interaction of treatment by bleeding day was significant ($P<.05$). These values can be found in appendix table 10. Again, the reasons for this may be environmental stress, or the stress associated with bleeding that to which ewes were subjected.

Treatment affected overall serum progesterone values as shown in table 14. Ewes in the ND treatment showed lower overall serum progesterone values ($P<.05$) followed by ewes in the ND+mel treatment. Ewes in the 8L:16D and 8L:16D+mel treatments had the highest serum progesterone levels ($P<.05$).

Serum progesterone level by week for all treatments combined differed ($P<.05$, appendix table 11). Weeks earlier in the experiment showed lower serum progesterone levels. As the experiment progressed, serum progesterone values increased. Week 8 to 13 were higher ($P<.05$) then wk 1 to 7. This would be expected because, as more ewes begin cycling and conceiving, serum progesterone levels will increase. The interaction of treatment by week was significant ($P<.05$). These values are shown in figures 9 to 12.

TABLE 14. LEAST SQUARES MEANS AND STANDARD ERRORS FOR OVERALL PROGESTERONE VALUES IN TRIAL 2

Treatment	Progesterone level, ng/ml
ND	1.0±.36[a]
8L:16D	1.8±.36[b]
ND+mel	1.5±.41[c]
8L:16D+mel	2.0±.36[b]

[a,b,c] Means with different superscripts differ (P<.05).

Ewes in the ND treatment had lower progesterone levels through most of the experiment than did the other three treatments, indicating fewer ewes cycling. Ewes in the 8L:16D, ND+mel, and 8L:16D+mel treatments reached and maintained serum progesterone levels above 1.0 ng/ml approximately 3 wk earlier than ewes in the ND treatment. The ewes in the ND treatment had an increase (P<.05) in serum progesterone at approximately wk 12. Ewes in the 8L:16D treatment showed this increase at wk 8, while ewes in the ND+mel and 8L:16D+mel treatments showed this increase at wk 7 and wk 9, respectively.

Figure 13 shows serum melatonin values across weeks for ewes in the four treatments. Treatment did not show serum melatonin differences (P>.05). Overall serum

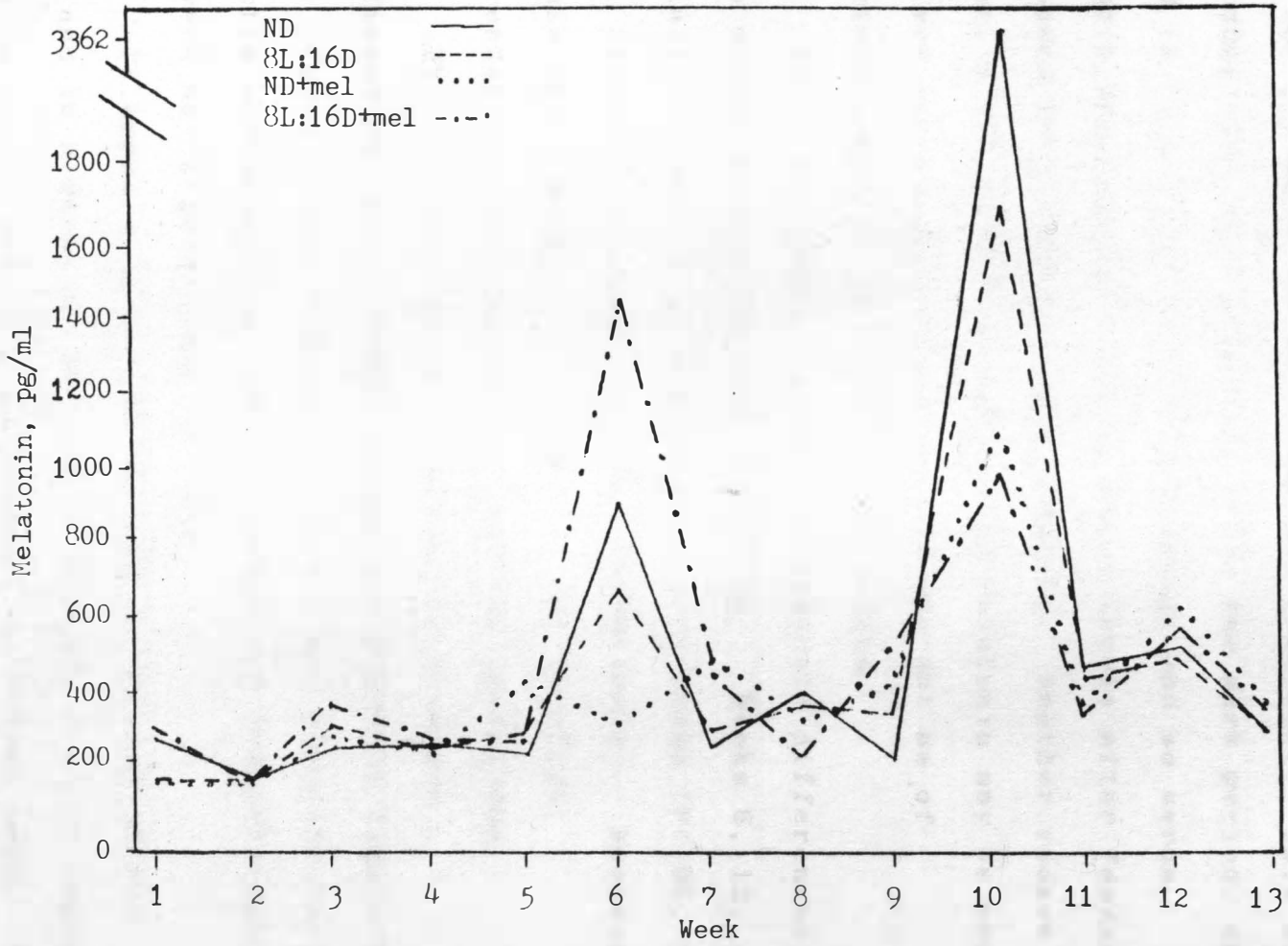


Figure 13. Melatonin levels by week for Trial 2 (S.E. for treatment within week range - 8.19 to 999.03, x = 202.1).

melatonin values were 586 pg/ml for the ewes in the ND treatment, 449 pg/ml for the ewes in the 8L:16D treatment, 412 pg/ml for the ewes in the ND+mel treatment, and 468 pg/ml for the ewes in the 8L:16D+mel treatment. This may be because ewes were bled soon after the dark period, which was 18 to 22 h after melatonin feeding, and so serum melatonin was probably back to basal levels after feeding and showed no difference from controls. Another reason may be that, although 3.5 mg per day of melatonin may be enough to effect reproductive function, it may not be of sufficient quantity to raise serum levels.

Serum melatonin values did indicate differences across weeks ($P<.05$, appendix table 12). Weeks 6, 12, and especially 10 were higher than all other weeks ($P<.05$, 847 pg/ml, 545 pg/ml and 1771 pg/ml, respectively). Reasons for this are unknown, but it may be due to slight differences in bleeding time or weather conditions.

The interaction of treatment by week was significant ($P<.05$). These values are shown in figure 13.

Correlations between prolactin and progesterone, prolactin and melatonin, and melatonin and progesterone by week were not signinficant ($P>.05$).

It appears that reduced light at a rate of 8 h light and 16 h dark, or feeding melatonin, or both appeared to hasten cyclic activity in normally anestrous ewes. This is shown by increased serum progesterone levels. This

reduced lighting schedule and(or) feeding melatonin also appears to reduce serum prolactin level, at least in ewes in long photoperiod (ND+mel). The 8L:16D treatment and feeding melatonin also appears to increase the number of ewes conceiving in the normal summer anestrous period.

SUMMARY

Trial 1 - Finnsheep x Targhee Ewes in Treatments 1-5

The effect of artificial photoperiods and(or) injecting 2-bromo-α-ergocryptine on stimulating the onset of estrus during the normal summer anestrous period was studied. Fifty Finnsheep x Targhee crossbred ewes, aged 2 to 5 yr, were randomly allotted within age to one of the following five treatment groups: 1) natural daylight (ND); 2) controlled light, 8 h light:16 h dark (8L:16D); 3) natural daylight plus 2.0 mg 2-bromo-α-ergocryptine injected two times per wk (ND+ergo); 4) controlled light, 8L:16D, plus 2.0 mg 2-bromo-α-ergocryptine injected two times per wk(8L:16D+ergo); and 5) controlled light, 16 h light:8 h dark, in which light was decreased 1 h per wk over an 8 wk period and then held at a constant 8L:16D for the remaining 7 wk (16L:8D-8L:16D). Ewes were continuously exposed to intact Suffolk rams to which a grease-paint mixture was applied twice daily to aid in detecting breeding activity.

There were no differences in number of breeding marks, days to first mark or days to second mark between treatments ($P>.05$).

Fewer days to lambing were observed in the ewes in treatments 1 and 3, probably due to the ewes not being anestrous at the time the trial started.

Ewes in the 8L:16D and 8L:16D+ergo treatments had more lambs ($P<.05$) than did ewes in the ND treatment (2.6 and 2.6 versus 1.6 lambs per ewe lambing, respectively). Ewes in treatment 2 also had a heavier average litter weight (11.2 kg) then did ewes in treatment 1 (7.3 kg, $P<.05$). More ewes lambed ($P<.05$) in treatments 2, 4 and 5 than in treatments 1 and 3 (80%, 100%, and 100% versus 50% and 50%, respectively).

Overall serum prolactin values showed treatment differences ($P<.05$), week differences ($P<.05$), and a significant interaction of treatment by week ($P<.05$). Serum prolactin values for the ewes in the 8L:16D treatment and in the ND+ergo treatment (166 ng/ml and 145 ng/ml, respectively) were higher ($P<.05$) than those ewes in the 16L:8D-8L:16D treatment (107 ng/ml). Ewes in treatment 1 (ND) showed the highest ($P<.05$) serum prolactin levels (246 ng/ml) and ewes in treatment 4 (8L:16D+ergo) had the lowest ($P<.05$) overall serum prolactin level (27 ng/ml).

Overall serum progesterone level differed between treatments ($P<.05$) and weeks ($P<.05$). The interaction of treatment by week was significant ($P<.05$). Ewes in treatment 1 and 5 did not differ ($P>.05$) in overall progesterone values (1.5 ng/ml and 1.2 ng/ml, respectively). Ewes in treatment 5 did differ ($P<.05$) from all other treatments. Treatment 2 ewes had higher ($P<.05$, 1.9 ng/ml) overall serum progesterone levels than did ewes

in treatments 1 and 5. Ewes in treatments 3 (1.8 ng/ml) and 4 (1.7 ng/ml) had higher overall progesterone levels ($P>.05$) than ewes in treatment 5.

It appears that reduced light will hasten cyclic activity and increase conception in the normally anestrous ewe. Ergocryptine appears to have no effect on initiating cyclic activity in anestrous ewes, although it will substantially decrease serum prolactin levels. A gradual increase in the length of the dark phase seems to have no additional benefits.

Trial 1 - Finnsheep x Targhee and Targhee Ewes in Treatments 1 and 2

This experiment involved the comparison of 20 straightbred 9-yr old Targhee (T) ewes and 20, 2 to 5-yr old Finnsheep x Targhee (FT) crossbred ewes in two treatments. These treatments were 1) natural daylight (ND), and 2) controlled lighting, 8 h light:16 h dark (8L:16D). These ewes were continuously exposed to intact Suffolk rams painted with a dye-colored grease to aid in detection of breeding activity.

There was a difference in starting weight ($P<.05$), probably due to breed effects. Ending weight and weight change throughout the experiment did not differ ($P>.05$).

There were no differences in number of marks or in days to first mark ($P>.05$).

Finnsheep x Targhee ewes in treatment 1 conceived an average of 38-39 d earlier than ewes of either breed in treatment 2 ($P<.05$), probably due to not all ewes being anestrous at the start of the experiment.

The number of ewes lambing in each treatment and breed group did not differ ($P>.05$). However, pooled ND treatment data showed fewer ewes lambing ($P<.10$) than did pooled 8L:16D data across breeds.

In treatments 1 and 2, FT ewes lambed 1.6 and 2.6 lambs per ewe lambing ($P<.05$). Targhee ewes differed from these values in respective treatments and from each other in the 2 treatments ($P<.05$). Targhee ewes in treatment 1 averaged 1.0 lambs per ewe lambing, and in treatment 2 averaged 1.6 lambs per ewe lambing. Litter weight differed between breeds ($P<.05$) for treatment 2 (FT = 11.2 kg, T = 9.0 kg), but not for treatment 1 (FT = 7.3 kg, T = 6.2 kg, $P>.05$). Litter weight in both treatments differed within breed ($P<.05$).

Treatment 2 ewes had overall prolactin levels of 136 ng/ml while treatment 1 ewes had overall prolactin levels of 266 ng/ml ($P<.05$). Week was also effected ($P<.05$) but with no specefic trend. Breed appeared to have no effect on prolactin level ($P>.05$).

Ewes in treatment 1 had an overall progesterone level of 1.3 ng/ml, while ewes in treatment 2 had an

overall progesterone level of 1.8 ng/ml. Treatment 2 ewes reached higher progesterone levels as time increased than did ewes in treatment 1 ($P<.05$). On the average, FT ewes had a higher progesterone level per wk than did T ewes ($P<.05$), probably due to an increased number of corpora lutea in the FT ewes because of greater ovulation rates.

There appears to be no difference in the reproductive performance of T and FT ewes in these treatments, except the differences caused by the FT ewes's natural ability to ovulate more, and conceive and carry more lambs.

Trial 2

The effect of artificial photoperiod and(or) feeding melatonin on stimulating the onset of estrus during the normal summer anestous period was studied. Sixty Finnsheep x Targhee crossbred ewes, aged 2 to 6 yr, were randomly allotted within age to one of four treatment groups: 1) natural daylight (ND); 2) controlled light, 8 h light:16 h dark (8L:16D); 3) natural daylight plus 3.5 mg melatonin fed per head daily (ND+mel); and 4) 8L:16D plus 3.5 mg fed per head daily (8L:16D+mel). Ewes were continuously exposed to intact Suffolk rams except during feeding. A grease-paint mixture was applied to the rams daily to aid in detecting breeding activity.

There was no difference in the number of breeding marks, or days to first mark between treatments ($P>.05$).

Forty percent of the ewes in treatment 1 lambed compared to an average of 95.2% lambing for the other three treatments combined ($P<.05$). The earliest average conception date and lambing date was July 26 and December 21 for the ewes in treatment 2. This was 10 d earlier ($P<.05$) than the conception or lambing date for ewes in treatment 1.

ND and ND+mel treated ewes averaged 1.5 and 1.8 lambs per ewe lambing, compared to 2.3 and 2.4 lambs per ewe lambing ($P<.05$) for ewes in treatment 2 and 4, respectively. Litter weight of lambs born per ewe lambing followed this same trend. Ewes in treatments 2 and 4 lambed an average litter weight of 10.3 kg, while ewes in treatments 1 and 3 lambed an average litter weight of 7.2 kg per ewe lambing ($P<.05$).

Serum prolactin and progesterone levels both differed for treatment and bleeding day ($P<.05$). The interaction of treatment by bleeding day was significant for both ($P<.05$). Ewes in treatment 1 had an overall serum prolactin level of 259 ng/ml, compared to 190 ng/ml for ewes in treatment 2, 191 ng/ml for ewes in treatment 3 and 196 ng/ml for ewes in treatment 4 ($P<.05$). Serum prolactin values for bleeding day with all treatments combined differed ($P<.05$), but with no specific trend except that

the first two bleeding days were higher ($P<.05$) than most other days.

Overall serum progesterone levels were 1.8 ng/ml and 2.0 ng/ml in treatments 2 and 4 ($P>.05$), compared to 1.5 ng/ml for ewes in treatment 3 ($P<.05$). Ewes in treatment 1 had the lowest ($P<.05$) overall serum progesterone level at 1.0 ng/ml. Weekly serum progesterone level with treatments combined showed higher levels ($P<.05$) in wk 8 to 13 than in wk 1 to 7. Ewes in treatment 1 showed increased ($P<.05$) serum progesterone levels at wk 12. Ewes in treatment 2 showed this increase ($P<.05$) at wk 8, while ewes in treatments 3 and 4 showed a similar increase ($P<.05$) at wk 7 and wk 9, respectively.

Overall serum melatonin values did not differ ($P>.05$) between treatments and ranged from 412 pg/ml to 586 pg/ml. Serum melatonin values did show week differences ($P<.05$) with wk 6, 10 and 12 being higher than all other weeks ($P<.05$). Reasons for this are unknown.

It appears that reduced light at a rate of 8L:16D, or feeding melatonin, or both hastened cyclic activity and increased conception in the normally anestrous ewe. Serum progesterone levels depict this. Feeding melatonin and(or) reduced light also appeared to lower serum prolactin levels in anestrous ewes. Serum melatonin levels were not affected in this experiment.

CONCLUSION

In Trial 1 shortened days increased reproductive efficiency in the normal summer anestrous period, although ergocryptine did not. Ergocryptine did, however, decrease serum prolactin levels dramatically. Changes in the light:dark cycle and feeding melatonin were used in Trial 2. Again, short days increased reproductive efficiency. Melatonin feeding also appeared to hasten cyclic activity and conception.

The results of these experiments are intriguing, but more research needs to be done to find a feasible and practicle method of extending the lambing season for producers. Future research may include such aspects as optimum melatonin level in the feed, feed processing with melatonin as an additive, and the mechanism of action of melatonin.

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APPENDIX

TABLE 1. LEAST SQUARES MEANS FOR PROLACTIN VALUES BY DAY OF BLEEDING FOR TRIAL 1, FT EWES IN TREATMENTS 1-5 (S.E.~30.7)

Day (date)	Prolactin, ng/ml
1 (5-16)	198
2 (5-19)	116
3 (5-23)	185
4 (5-26)	178
5 (5-30)	62
6 (6- 2)	89
7 (6- 6)	68
8 (6- 9)	92
9 (6-13)	70
10 (6-16)	51
11 (6-20)	52
12 (6-23)	173
13 (6-27)	35
14 (6-30)	25
15 (7- 4)	97
16 (7- 7)	135
17 (7-11)	96
18 (7-14)	125
19 (7-18)	129
20 (7-21)	160
21 (7-25)	182
22 (7-28)	195
23 (8- 1)	92
24 (8- 4)	143
25 (8- 8)	235
26 (8-11)	139
27 (8-15)	336
28 (8-18)	124
29 (8-22)	326
30 (8-25)	244

TABLE 2. LEAST SQUARES MEANS AND STANDARD ERRORS FOR THE INTERACTION OF TREATMENT BY DAY OF BLEEDING FOR PROLACTIN IN TRIAL 1, FT EWES IN TREATMENTS 1-5

	Prolactin, ng/ml				
	Treatments**				
Day*	1	2	3	4	5
1	14± 4.2	514±310.1	20± 2.8	119± 35.9	326±143.1
2	27± 8.5	24± 4.2	19± 3.6	92± 17.9	417±157.5
3	82± 25.5	142± 66.8	44± 6.8	158± 30.0	497±150.4
4	196± 39.4	259± 81.8	96± 15.5	179±159.6	161± 37.5
5	66± 26.0	37± 7.6	23± 9.6	2± 1.0	181± 56.7
6	197± 64.1	93± 10.0	64± 6.7	1± .8	92± 30.9
7	71± 7.6	90± 12.1	77± 6.1	16± 5.2	86± 14.3
8	152± 56.7	155± 92.6	92± 18.7	8± 1.8	54± 9.5
9	92± 5.7	75± 17.0	88± 5.2	27± 10.6	66± 5.2
10	109± 18.4	51± 18.8	28± 5.7	2± .7	62± 19.2
11	104± 24.9	23± 7.7	99± 24.3	10± 5.3	25± 5.9
12	235± 61.1	186± 87.5	320±118.1	25± 6.9	99± 32.4
13	101± 27.0	12± 5.7	48± 4.2	4± .5	11± 2.8
14	56± 13.6	13± 1.3	46± 18.9	2± .5	8± 1.8
15	131± 18.4	150± 69.4	135± 18.6	14± 3.1	56± 11.6
16	281± 28.8	109± 32.5	177± 51.5	38± 31.6	69± 20.1
17	112± 8.9	149± 27.6	150± 33.2	20± 5.7	47± 8.8
18	385±117.8	94± 30.6	102± 32.5	6± 1.5	40± 14.4
19	293± 53.4	80± 21.4	236± 96.0	6± 2.5	29± 6.6
20	305± 59.3	78± 21.3	378±296.7	3± 1.2	35± 7.2
21	425± 92.4	132± 31.7	257± 58.5	13± 3.1	83± 25.4
22	550±130.6	115± 27.6	178±106.7	11± 2.6	118± 33.3
23	180± 28.7	69± 25.8	116± 37.3	20± 12.3	77± 20.1
24	394± 92.0	116± 20.5	123± 62.6	4± .9	78± 13.7
25	753±417.3	288±114.4	53± 23.3	3± 1.1	80± 19.0
26	342±104.6	102± 23.9	116± 53.9	3± .9	134± 44.1
27	394±137.8	502±205.9	715±475.5	0± 0	69± 20.2
28	193± 65.6	268± 70.6	86± 32.2	8± 1.0	66± 16.9
29	420±159.0	757±450.0	409±247.2	0± 0	43± 12.4
30	740±295.7	314±115.4	71± 28.6	2± .8	94± 18.5

*See appendix table 1 for corresponding dates.
**See page 20 for corresponding treatments.

TABLE 3. LEAST SQUARES MEANS FOR PROGESTERONE VALUES BY WEEK FOR TRIAL 1, FT EWES IN TREATMENTS 1-5 (S.E.~.211)

Week (date)	Progesterone, ng/ml
1 (5-19)	.39
2 (5-26)	.97
3 (6- 2)	.86
4 (6- 9)	.34
5 (6-16)	.79
6 (6-23)	.94
7 (6-30)	.79
8 (7- 7)	1.16
9 (7-14)	1.21
10 (7-21)	2.03
11 (7-28)	2.26
12 (8- 4)	1.79
13 (8-11)	2.41
14 (8-18)	3.44
15 (8-25)	4.75

TABLE 4. LEAST SQUARES MEANS FOR PROLACTIN VALUES BY DAY OF BLEEDING FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2 (S.E.~48.4)

Day*	Prolactin, ng/ml
1	176
2	82
3	200
4	192
5	147
6	193
7	209
8	131
9	207
10	296
11	157
12	434
13	160
14	238
15	4
16	176
17	82
18	200
19	192
20	147
21	193
22	209
23	131
24	207
25	296
26	157
27	434
28	160
29	238
30	376

*See appendix table 1 for corresponding dates.

TABLE 5. LEAST SQUARES MEANS FOR THE INTERACTION OF TREATMENT BY DAY OF BLEEDING FOR PROLACTIN IN TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2 (S.E.~68.4)

	Prolactin, ng/ml	
	Treatment	
Day*	ND	8L:16D
1	142	210
2	82	81
3	239	161
4	231	153
5	227	67
6	272	115
7	319	99
8	170	93
9	320	95
10	460	133
11	224	89
12	615	254
13	173	148
14	237	239
15	6	2
16	142	210
17	82	81
18	239	161
19	231	153
20	227	67
21	272	115
22	319	99
23	170	93
24	319	95
25	459	133
26	224	89
27	615	254
28	173	148
29	237	239
30	565	187

*See appendix table 1 for corresponding dates.

TABLE 6. LEAST SQUARES MEANS FOR THE INTERACTION OF TREATMENT BY BREED FOR PROLACTIN IN TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2 (S.E.~17.7)

Treatment	Breed	Prolactin, ng/ml
ND	T	300
ND	FT	232
8L:16D	T	112
8L:16D	FT	159

TABLE 7. LEAST SQUARES MEANS FOR PROGESTERONE VALUES BY WEEKS FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2 (S.E.~.219)

Week*	Progesterone, ng/ml
1	.15
2	1.47
3	.61
4	.34
5	.93
6	1.11
7	.78
8	1.24
9	1.43
10	1.93
11	2.27
12	1.70
13	2.03
14	3.14
15	4.24

*See appendix table 3 for corresponding dates.

TABLE 8. LEAST SQUARES MEANS FOR THE INTERACTION OF WEEK BY BREED FOR PROGESTERONE VALUES FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2 (S.E.~.310)

Week*	Breed	Progesterone, ng/ml
1	T	.07
1	FT	.23
2	T	1.61
2	FT	1.32
3	T	.38
3	FT	.85
4	T	.37
4	FT	.31
5	T	.95
5	FT	.91
6	T	1.04
6	FT	1.18
7	T	.63
7	FT	.92
8	T	1.12
8	FT	1.37
9	T	1.36
9	FT	1.50
10	T	1.66
10	FT	2.20
11	T	2.09
11	FT	2.46
12	T	1.61
12	FT	1.80
13	T	2.01
13	FT	2.05
14	T	2.94
14	FT	3.35
15	T	3.85
15	FT	4.63

*See appendix table 3 for corresponding dates.

TABLE 9. LEAST SQUARES MEANS FOR PROLACTIN VALUES BY DAY OF BLEEDING FOR TRIAL 2 (S.E.~48.1)

Day (date)	Prolactin, ng/ml
1 (6- 1)	403
2 (6- 5)	349
3 (6- 8)	179
4 (6-12)	326
5 (6-15)	199
6 (6-19)	223
7 (6-22)	250
8 (6-26)	162
9 (6-29)	147
10 (7- 3)	185
11 (7- 6)	172
12 (7-10)	175
13 (7-13)	202
14 (7-17)	153
15 (7-20)	319
16 (7-24)	179
17 (7-27)	93
18 (7-31)	103
19 (8- 3)	181
20 (8- 7)	314
21 (8-10)	175
22 (8-14)	172
23 (8-17)	168
24 (8-21)	233
25 (8-24)	163

TABLE 10. LEAST SQUARES MEANS AND STANDARD ERRORS FOR THE INTERACTION OF TREATMENT BY DAY OF BLEEDING FOR TRIAL 2

	Prolactin, ng/ml			
	Treatments**			
Day*	1	2	3	4
1	244± 45.8	754±255.2	300±122.5	314± 65.8
2	289± 45.7	316± 47.5	319± 54.4	471±255.5
3	138± 27.9	142± 21.9	188± 92.4	247± 68.0
4	303± 44.5	347± 99.3	216± 39.6	438±130.4
5	195± 32.0	149± 36.3	199± 82.2	255± 74.6
6	265± 43.4	306± 80.9	89± 22.4	233± 49.6
7	190± 22.5	245± 50.0	323±128.5	242± 71.0
8	344± 69.2	90± 19.4	100± 20.4	115± 27.9
9	229± 34.6	125± 32.6	71± 14.4	161± 54.3
10	291± 74.2	317±247.3	27± 4.6	107± 21.7
11	245± 31.0	114± 21.6	105± 31.3	225± 48.4
12	206± 32.0	189± 18.8	71± 6.8	233± 71.6
13	301± 53.2	232± 40.5	53± 11.2	224± 50.3
14	115± 13.1	88± 37.4	36± 17.8	372±260.7
15	241± 74.6	113± 41.2	773±334.8	147± 71.4
16	384± 59.4	52± 10.4	88± 32.4	194±131.4
17	172± 40.1	36± 5.6	48± 16.4	117± 64.5
18	173± 14.8	50± 10.0	142± 56.0	48± 17.4
19	309± 59.1	143± 80.8	129± 38.2	145± 38.5
20	288± 56.2	380±261.1	493±301.1	95± 28.0
21	256± 94.3	131± 38.3	80± 12.4	230± 84.5
22	470±255.6	52± 10.2	142± 21.5	23± 4.5
23	408±111.6	79± 17.7	139± 21.7	48± 9.4
24	223± 58.0	181± 56.1	451±323.0	75± 11.8
25	189± 26.8	133± 28.6	194± 52.3	134± 42.5

*See appendix table 9 for corresponding dates.
**See page 26 for corresponding treatments.

TABLE 11. LEAST SQUARES MEANS FOR PROGESTERONE VALUES BY WEEK FOR TRIAL 2 (S.E.~.187)

Week (date)	Progesterone, ng/ml
1 (6- 1)	1.07
2 (6- 8)	.90
3 (6-15)	.77
4 (6-22)	.63
5 (6-29)	.31
6 (7- 7)	.45
7 (7-14)	.72
8 (7-21)	1.61
9 (7-28)	2.51
10 (8- 3)	1.76
11 (8-10)	3.08
12 (8-17)	3.29
13 (8-24)	3.58

TABLE 12. LEAST SQUARES MEANS FOR MELATONIN VALUES BY WEEK FOR TRIAL 2 (S.E.~101.5)

Week*	Melatonin, pg/ml
1	224
2	172
3	295
4	253
5	323
6	847
7	378
8	336
9	378
10	1771
11	396
12	545
13	307

*See appendix table 11 for corresponding dates.

TABLE 13. LEAST SQUARES ANALYSIS OF VARIANCE FOR STARTING EWE WEIGHT FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	557.09	139.27	1.40
Error	45	4482.25	99.61	
Total	49	5039.34		

TABLE 14. LEAST SQUARES ANALYSIS OF VARIANCE FOR ENDING EWE WEIGHT FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	1389.56	347.39	3.04*
Error	45	5134.34	114.10	
Total	49	6523.90		

*P<.05.

TABLE 15. LEAST SQUARES ANALYSIS OF VARIANCE FOR EWE WEIGHT CHANGE FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	560.68	140.17	5.82*
Error	45	1083.49	24.08	
Total	49	1644.17		

*P<.05.

TABLE 16. LEAST SQUARES ANALYSIS OF VARIANCE FOR NUMBER OF BREEDING MARKS FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	2.92	.73	.53
Error	45	62.20	1.38	
Total	49	65.10		

TABLE 17. LEAST SQUARES ANALYSIS OF VARIANCE FOR DAYS TO FIRST MARK FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	6249.99	1562.50	2.36
Error	37	24503.15	662.25	
Total	41	30753.14		

TABLE 18. LEAST SQUARES ANALYSIS OF VARIANCE FOR DAYS TO SECOND MARK FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	1860.18	465.04	.59
Error	24	19003.96	791.83	
Total	28	20864.14		

TABLE 19. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBING DATE FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	13873.13	3468.28	9.44*
Error	33	12130.38	367.59	
Total	37	26003.50		

*P<.0001.

TABLE 20. CHI SQUARE ANALYSIS FOR NUMBER OF EWES LAMBING PER TREATMENT GROUP IN TRIAL 1, FT EWES IN TREATMENTS 1-5

Treatment	Observed, n	Expected, n
ND	5	7.60
8L:16D	8	7.60
ND+ergo	5	7.60
8L:16D+ergo	10	7.60
16L:8D-8L:16D	10	7.60

Experimental chi square values = 13.83; P<.05. Chi square value = 9.49 for four degrees of freedom.

TABLE 21. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBS PER EWE LAMBING FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	4.74	1.19	2.29*
Error	33	17.08	.52	
Total	37	21.82		

*P<.05.

TABLE 22. LEAST SQUARES ANALYSIS OF VARIANCE FOR LITTER WEIGHT OF LAMBS BORN PER EWE LAMBING IN TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	523.40	130.85	1.96*
Error	33	2205.22	66.83	
Total	37	2728.62		

*P<.05.

TABLE 23. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROLACTIN FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	7815470.71	1953867.70	21.75*
Days	29	8718122.99	300624.93	3.35*
Trt*Day	116	21186925.17	182645.91	2.03*
Error	1350	121289013.30	89843.71	
Total	1499	159009532.17		

*P<.001.

TABLE 24. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROGESTERONE VALUES FOR TRIAL 1, FT EWES IN TREATMENTS 1-5

Source of variation	DF	SS	MS	F
Treatment	4	39.03	9.76	4.34*
Week	14	1036.69	74.05	32.96**
Trt*Week	56	149.01	2.66	1.18
Error	675	1516.32	2.25	
Total	749	2471.05		

*P<.0018, **P<.0001.

TABLE 25. LEAST SQUARES ANALYSIS OF VARIANCE FOR STARTING EWE WEIGHT FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	282.83	282.83	4.09*
Breed	1	483.66	483.66	6.99**
Trt*Brd	1	.33	.33	0
Error	36	2490.70	69.19	
Total	39	3257.52		

*P<.05, **P<.01.

TABLE 26. LEAST SQUARES ANALYSIS OF VARIANCE FOR ENDING EWE WEIGHT FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	7.46	7.46	.10
Breed	1	370.99	370.99	5.22*
Trt*Brd	1	13.97	13.97	.20
Error	36	2557.56	71.04	
Total	39	2949.98		

*P<.03.

TABLE 27. LEAST SQUARES ANALYSIS OF VARIANCE FOR WEIGHT CHANGE FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	382.15	382.15	17.06*
Breed	1	7.45	7.45	.33
Trt*Brd	1	10.00	10.00	.45
Error	36	806.49	22.40	
Total	39	1207.09		

*P<.0002.

TABLE 28. LEAST SQUARES ANALYSIS OF VARIANCE FOR NUMBER OF BREEDING MARKS FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	.90	.90	.75
Breed	1	3.60	3.60	2.99
Trt*Brd	1	0	0	0
Error	36	43.40	1.21	
Total	39	47.90		

TABLE 29. LEAST SQUARES ANALYSIS OF VARIANCE FOR DAYS TO FIRST MARK FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	114.21	114.21	.11
Breed	1	586.73	586.73	.57
Trt*Brd	1	1501.73	1501.73	1.46
Error	27	27813.45	1030.13	
Total	30	30093.87		

TABLE 30. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBING DATE FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	4331.27	4331.27	6.18*
Breed	1	736.49	736.49	1.05
Trt*Brd	1	938.02	938.02	1.34
Error	25	17524.01	2001.93	
Total	28	23529.79		

*P<.02.

TABLE 31. CHI SQUARE ANALYSIS FOR NUMBER OF EWES LAMBING PER TREATMENT AND BREED GROUP FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Treatment	Breed	Observed, n	Expected, n
ND	T	6	7.25
ND	FT	5	7.25
8L:16D	T	10	7.25
8L:16D	FT	8	7.25

Experimental chi square value = 7.30, P>.05. Chi square value = 7.81 for three degrees of freedom.

TABLE 32. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBS PER EWE LAMBING IN TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	4.18	4.18	19.11*
Breed	1	5.35	5.35	24.41**
Trt*Brd	1	.31	.31	1.39
Error	25	5.48	.22	
Total	28	15.31		

*P<.0002, **P<.0001.

TABLE 33. LEAST SQUARES ANALYSIS OF VARIANCE FOR LITTER WEIGHT OF LAMBS BORN PER EWE LAMBING FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	663.07	663.07	14.78**
Breed	1	225.91	225.91	5.03*
Trt*Brd	1	19.84	19.84	.44
Error	24	898.82	37.44	
Total	27	1807.64		

**P<.0008, *P<.03.

TABLE 34. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROLACTIN FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	10250326.58	10250326.58	54.79***
Day	29	20186419.25	696083.41	3.72***
Trt*Day	29	9659492.77	333085.96	1.78*
Breed	1	67809.73	67809.73	.36
Trt*Brd	1	1970828.44	1970828.44	10.53**
Day*Brd	29	3784897.94	130513.72	.70
Trt*Day*Brd	29	6570751.59	226577.64	1.21
Error	2280	426546486.68	187081.79	
Total	2399	479037012.98		

*P<.0065, **P<.0012, ***P<.0001.

TABLE 35. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROGESTERONE VALUES FOR TRIAL 1, T AND FT EWES IN TREATMENTS 1 AND 2

Source of variation	DF	SS	MS	F
Treatment	1	38.05	38.05	19.85**
Week	14	651.56	46.54	24.28**
Trt*Week	14	122.54	8.75	4.57**
Breed	1	7.68	7.68	4.01*
Trt*Brd	1	1.49	1.49	.78
Week*Brd	14	9.91	.71	.37
Trt*Week*Brd	14	6.05	.43	.23
Error	540	1035.16	1.92	
Total	599	1872.45		

**P<.0001, *P<.05.

TABLE 36. LEAST SQUARES ANALYSIS OF VARIANCE FOR STARTING EWE WEIGHT FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	328.28	109.43	1.33
Error	53	4371.90	82.49	
Total	56	4700.18		

TABLE 37. LEAST SQUARES ANALYSIS OF VARIANCE FOR ENDING EWE WEIGHT FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	321.69	107.23	1.09
Error	53	5195.97	98.04	
Total	56	5517.67		

TABLE 38. LEAST SQUARES ANALYSIS OF VARIANCE FOR EWE WEIGHT CHANGE FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	50.36	16.79	.66
Error	53	1355.06	25.57	
Total	56	1405.42		

TABLE 39. LEAST SQUARES ANALYSIS OF VARIANCE FOR NUMBER OF BREEDING MARKS FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	4.13	1.38	1.32
Error	53	55.45	1.05	
Total	56	59.58		

TABLE 40. LEAST SQUARES ANALYSIS OF VARIANCE FOR DAYS TO FIRST MARK FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	1630.08	543.36	2.40
Error	53	12023.85	226.87	
Total	56	13653.93		

TABLE 41. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBING DATE FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	695.92	231.97	2.98*
Error	42	3265.56	77.75	
Total	45	3961.48		

*P<.05.

TABLE 42. CHI SQUARE ANALYSIS FOR NUMBER OF EWES LAMBING PER TREATMENT GROUP FOR TRIAL 2

Treatment	Observed, n	Expected, n
ND	6	12.10
8L:16D	15	12.10
ND+mel	11	9.68
8L:16D+mel	14	12.10

Experimental chi square value = 21.28, P<.05. Chi square value = 7.81 for three degrees of freedom.

TABLE 43. LEAST SQUARES ANALYSIS OF VARIANCE FOR LAMBS PER EWE LAMBING IN TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	52920.63	17640.21	5.71*
Error	42	129765.11	3089.65	
Total	45	182685.74		

*P<.002.

TABLE 44. LEAST SQUARES ANALYSIS OF VARIANCE FOR LITTER WEIGHT OF LAMBS PER EWE LAMBING IN TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	1093.40	364.47	5.71*
Error	42	2681.10	63.84	
Total	45	3774.50		

*P<.002.

TABLE 45. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROLACTIN FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	1219455.60	406485.20	3.12*
Day	24	8123556.32	338481.51	2.56**
Trt*Day	72	16858090.24	234140.14	1.79**
Error	1325	172863082.88	130462.72	
Total	1424	198677315.04		

*P<.03, **P<.001.

TABLE 46. LEAST SQUARES ANALYSIS OF VARIANCE FOR PROGESTERONE FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	124.77	41.59	21.00*
Week	12	900.74	75.06	37.89*
Trt*Week	36	259.88	7.22	3.64*
Error	689	1364.81	1.98	
Total	740	2643.39		

*P<.0001.

TABLE 47. LEAST SQUARES ANALYSIS OF VARIANCE FOR MELATONIN FOR TRIAL 2

Source of variation	DF	SS	MS	F
Treatment	3	1102061.42	367353.80	1.78
Week	12	43119506.32	1197764.10	17.43*
Trt*Week	36	21604585.61	600127.36	2.91*
Error	208	42891660.40	206209.91	
Total	259	108717813.77		

*P<.0001.